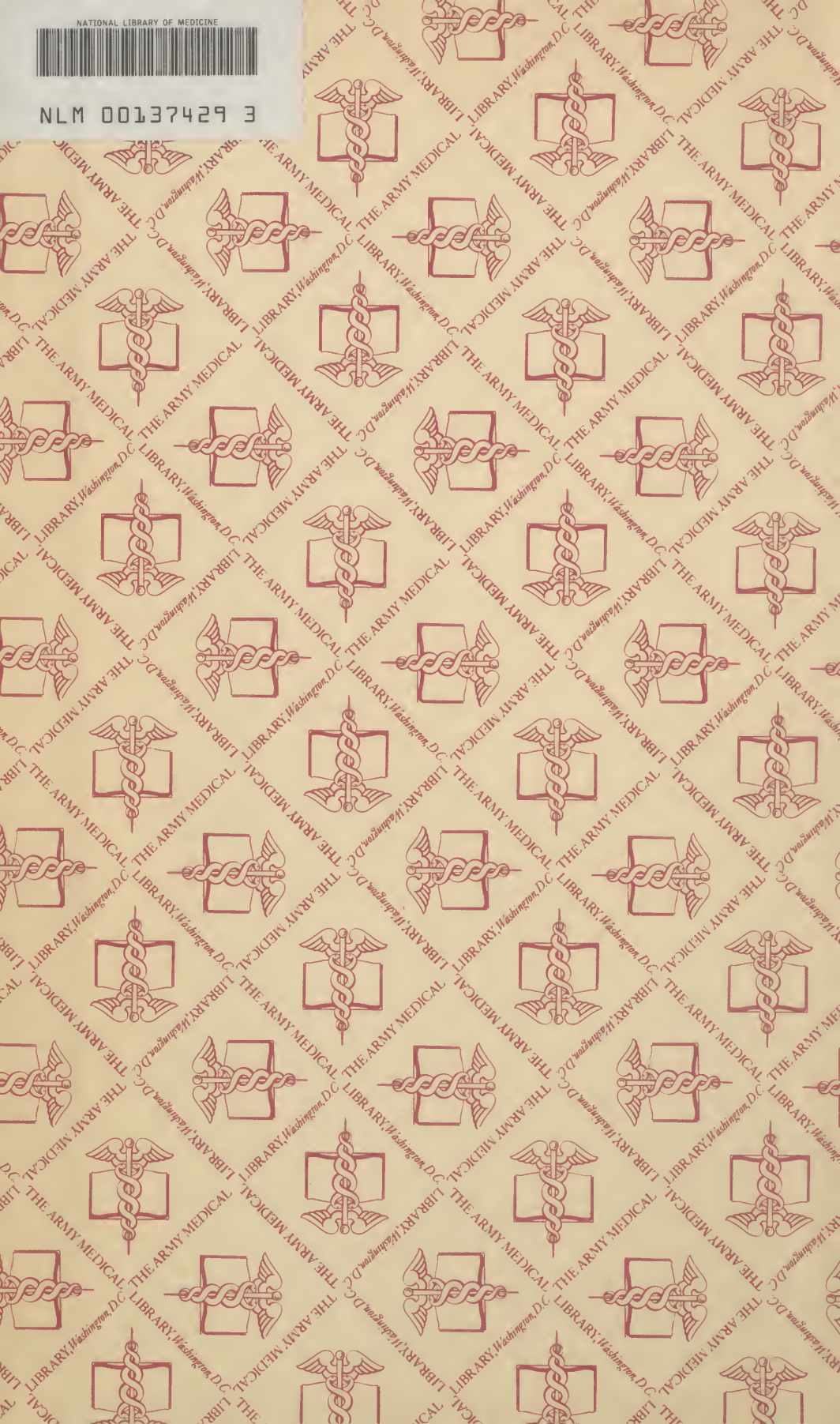


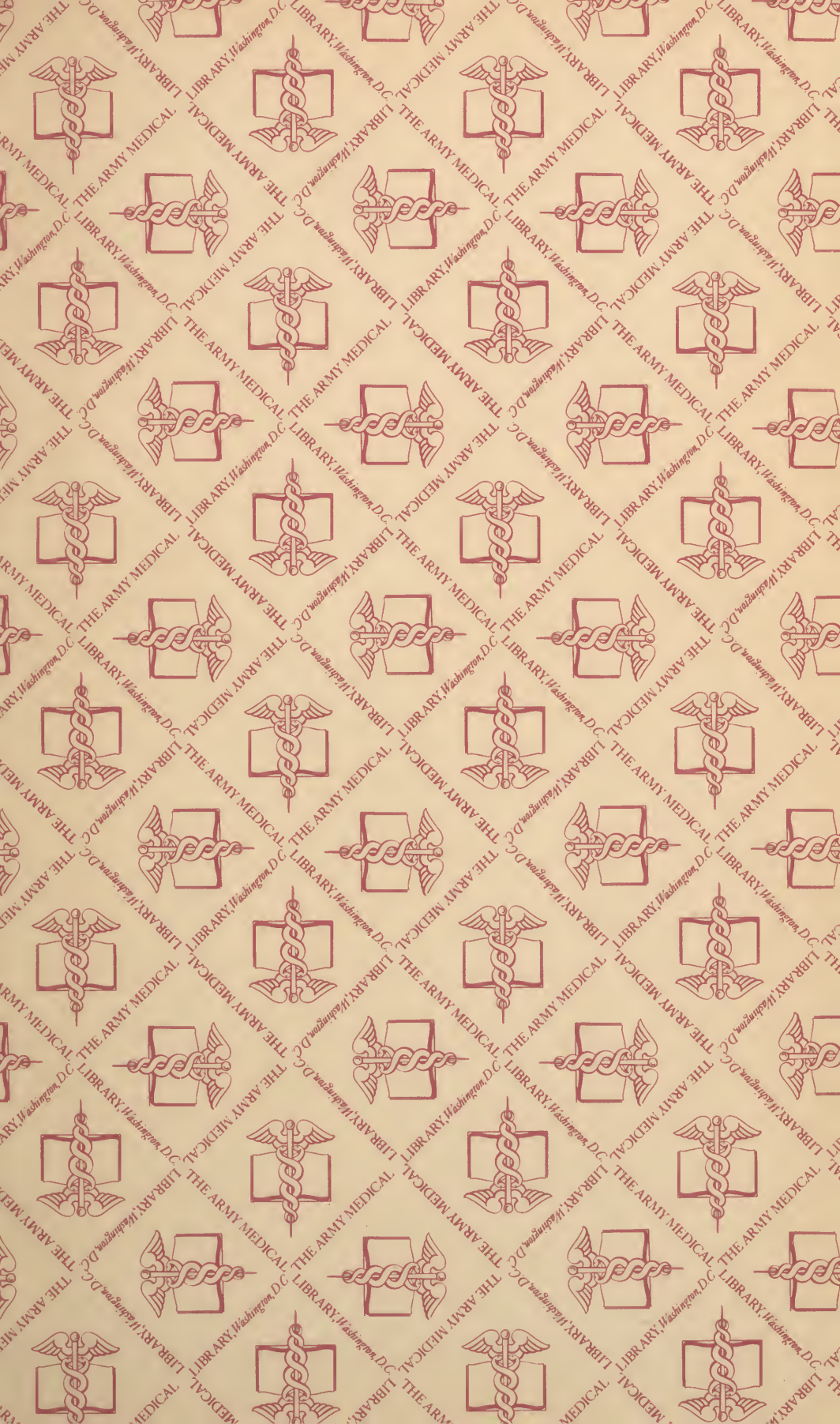




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THE  
PRACTICE OF MEDICINE

BY

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WITH SECTIONS ON

DISEASES OF THE NERVOUS SYSTEM

BY

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VOLUME I.

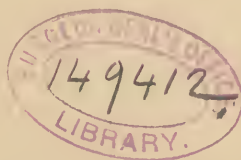
SPECIFIC INFECTIOUS DISEASES AND DISEASES OF THE  
NERVOUS SYSTEM.

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## PREFACE.

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In the preparation of this work the author has endeavored to write from a thoroughly practical standpoint. That there is a place in Homœopathic literature for such a treatise will, it is believed, be generally admitted. In the therapeutic sections the aim has been to consider the relationship of medicines to disease conditions, especially from the supreme position of clinical experience, which must always prove the ultimate test of the value of any remedy. This treatment of the subject avoids certain objectionable methods of the past, particularly the introduction of large numbers of symptoms from the *materia medica*, upon theoretical grounds only, and the failure to indicate the relative clinical value of medicines. The sections upon therapeutics contained in a work upon the Practice of Medicine are not of sufficient magnitude to permit of the inclusion of extensive discussions in special therapeutics, or the introduction of the *materia medica* bodily. Auxiliary methods of treatment have not been omitted, since a complete guide to practice must include all important therapeutic measures, even if infrequently employed.

The preparation of the section treating of Diseases of the Nervous System has been committed to Dr. Clarence Bartlett, who has undoubtedly presented the subject more satisfactorily than is possible for one whose studies embrace the domain of general medicine. Dr. Bartlett has also rendered most valuable assistance in the collection and elaboration of material for other portions of the work. The article upon Syphilis is entirely from his pen.

Acknowledgments are also due Dr. Claude R. Norton for aid in the reading of proof and for many valuable suggestions; to Dr. Edward R. Snader for a variety of services in the preparation of the section upon the Diseases of the Chest; and to Dr. W. D. Bayley for assistance in revising the manuscript and correcting the proof of the section on Nervous Diseases. To my colleague, Dr. Charles M. Thomas, I am indebted for most valuable criticisms which have greatly enhanced the value of the work.

#### ERRATA.

Page 80, line 21 from the top, "administer" instead of "adminster."

Page 353, line 6 from the top, "officious" instead of "efficacious."

Page 379, line 3 from the top, "morning" instead of "evening."

Page 392, line 19 from the top, insert "three times" after "for one hour."

Page 406, line 11 from the top, "those" instead of "these."



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# PRACTICE OF MEDICINE.

## GENERAL CONSIDERATIONS CONCERNING THE SPECIFIC INFECTIOUS DISEASES.

This extensive group of affections, characterized by the quality of communicability from one member of the human family to another, and in some instances from man to animals, or from animals to man, has been considerably enlarged during recent years by the addition of a number of diseases, the dependence of which upon specific infectious causes had hitherto been unknown. Each member possesses a peculiar individuality, and all are etiologically dependent upon pathogenic micro-organisms.

The acute infectious diseases prevail endemically or epidemically, and by this is meant respectively that they tend to persist in certain regions of country, being there favored by local conditions; or that their development and spread are rapid, but their continuance of rather short duration.

A study of the acute infectious group of diseases is fitly preceded by a review of the subjects of fever, contagion, immunity, prophylaxis and other matters bearing intimately upon infectious disease. All of these subjects have been studied with increasing zeal during the past decade, with the result of making most important additions to our knowledge.

### FEVER.

The words fever and pyrexia do not possess exactly the same significance; pyrexia being applied to simple abstract elevation of temperature; fever to elevation of temperature *plus* the group of symptoms which are so constantly associated with increase of heat. Cases of so-called fever, even fatal ones, may exist with little or no elevation of temperature. This has been repeatedly observed, especially in typhoid fever. The temperature of the healthy human body is marvellously stationary under most opposite conditions, being nearly the same, whether in the frigid or torrid zone. We are accustomed to stating it as 98.4° F., which in this

region, at least, is seldom departed from more than a few tenths of a degree, although the books allow of a daily fluctuation of a degree or more as compatible with health. Many instances upon which statements as to marked diurnal fluctuations are based, are, however, in the estimation of the writer, pathological, and therefore such pronounced variations should always incite us to careful clinical investigations lest concealed phenomena escape observation. Cases of incipient or latent plithisis probably furnish the largest number of apparently healthy persons with a fluctuating temperature. The daily variation, whether slight or very decided, is represented by the lowest temperature in the morning after about 2 A.M., with a rise to the normal during the early forenoon, reaching the maximum some time between noon and late evening. Exercise, if of a violent character, raises the temperature somewhat, while protracted mental effort may lower it. A cold atmosphere depresses it, while a hot one, by diminishing radiation, elevates it. In the tropics it is not uncommon to find an elevation of the temperature of a degree or rather more. The temperature is also higher in the young (and often in the old) than in the middle-aged. Elevations of temperature not to be accounted for by the preceding statements, or in excess of what we are justified in considering as physiological, are pathological in character, and usually accompanied by well-defined symptoms of disturbance in health. Temperatures ranging from normal to 99.5° or 100° F. are frequently referred to as sub-febrile; between 100° and 102° or 103° as moderate fever; 104° to 105° as high fever; and above the latter point, as hyperpyrexia. This division can be approximate only.

Depressed temperatures are called subnormal if not below 96° F.; but if below this point, symptoms of failing circulation are usually developed and the temperature is rated as collapsic. A fever attack may be divided into several stages: (1) A stage of invasion, which may be preceded by one of prodromes. (2) A stage of acme or the fastigium. (3) The stage of defervescence or decline. The more rapid in its evolution the paroxysm of fever, the more characteristic will be these several stages. They are best seen in an attack of malarial intermittent fever. While in the *first stage* the patient complains of being chilly, and there are all the external evidences of a chill, the thermometer indicates a rise of temperature from the very beginning. The external coldness and paleness, and in some instances the shrivelled, cyanotic appearance, are due to spasm of the superficial vessels. They are most conspicuous in the extremities, also in the ear, lips and nose. During this stage the finger, but better the sphygmograph, will indicate a high arterial tension. Even with the finger the pulse may be easily observed to be hard, prolonged, and of small calibre. Headache may be marked, and the patient apprehensive. After a period varying from minutes to hours, the shivering ceases, the surface of the body becomes warm, the pulse fuller and bounding, the

temperature rises, the headache and general pains increase or decrease, in fact the general well-known symptoms of fever appear and the second stage, of acme or fastigium, has been established. The third stage (defervescence) presents greater variety. Usually there is a gradual fall of the temperature, to or below the normal point, not accompanied by any notable events, such a method being designated as a *termination by lysis*. A rapid fall of temperature, which may be accompanied by a profuse urination, diarrhœa, sweat, collapse, etc., is termed a *termination by crisis*. These methods may be variously combined.

**Symptoms of Fever.**—Aside from the rise in temperature there are certain phenomena quite constantly associated with the febrile state. These will be briefly considered.

**CIRCULATORY.** The pulse is generally accelerated. In moderate fever it varies from 100 to 110, with increasing temperature it may rise to 120, 130, 160, or more. Statements of a certain ratio existing between the frequency of the pulse and the rise of the temperature have been made, but there are so many exceptions that they are of little value. The pulse is usually full and bounding, or hard and wiry, in the early stage of fever; becoming weak, more frequent and perhaps disturbed in rhythm or intermittent with the progress of long-continued or low types of fever. The heart's impulse at the same time grows feebler and the first sound loses its muscular quality, becoming valvular in character. Coincident blood-changes are usually in the direction of a darker color and a greater fluidity. The salts are reduced in quantity and with the progress of the fever the albumen is diminished as well as the red blood corpuscles. The fibrin may be increased. With failing circulation the surface of the body may become cool, clammy, or cyanotic.

**RESPIRATION.** The respirations are increased. In young subjects, and in certain diseases, as pneumonic fever, the rate of increase may be great. Rates of 40, 60, or 80, are not uncommon. It must be remembered, however, that in many affections the respiration is influenced by causes other than the fever. The elimination of carbonic acid is usually largely increased. If disorders of rhythm are present we must look to causes other than fever.

**GASTRO-INTESTINAL TRACT.** There is usually a loss of appetite, and nausea and vomiting may occur, especially in the initial stage of eruptive fevers. If persistent, it is frequently secondary to a kidney complication. The bowels are usually constipated.

**TONGUE.** The tongue is quickly furred, and in protracted fevers becomes dry and may be denuded of its epithelium; dryness usually first appears at the centre. The exfoliation of epithelium is first apparent along the edges of the organ and upon a central longitudinal stripe. The organ may become red, raw, tender, or cracked.

**GENITO-URINARY TRACT.** The urine is generally lessened in quan-

tity and correspondingly heightened in color. It is strongly acid in reaction. The odor may be increased. The solids, especially the urea and uric acid, per ounce, are generally much increased, but the scantiness of the secretion may make the total excretion of solids for the twenty-four hours less than normal. With the cooling of the urine, urates, and often uric acid, are precipitated. Chlorides are generally reduced, indeed may disappear. Albumen is frequently present in the urine in minute quantities, and in large quantities especially in diphtheria. The concentrated acid urine is passed frequently, or may be retained.

**THE NERVOUS SYSTEM.** Symptoms of disturbance of the nervous system are usually present. Early there are headache, pains in the back and limbs, and a general sense of languor and indisposition, prostration may be pronounced in the early stage. Distressing restlessness, sleeplessness, and various grades of delirium are common in the more protracted cases of fever. Tremors, subsultus tendinum, picking of the bed-clothes, grotesque motions of the arms, maniacal delirium, convulsions, and coma are met in the typhoidal forms of fever.

Various types of fever are recognized. These are determined by the temperature range, and the character, combination and progress of the symptoms. The most important are the following:

**CONTINUED FEVER.** In the case of the continuance of a fever for several days, during which the daily remissions are not greater than one to two degrees, it is termed a continued fever. The affections representing this group run quite a regular course. It embraces most members of the acute infectious group. Defervescence may be rapid (*crisis*) or gradual (*lysis*).

**REMITTING FEVER** is characterized by greater daily remissions than occur in continued fever. It is not unusual for a continued form of fever to become remitting in character during decline. This is common during the third week of typhoid fever. With the remission of the fever there is usually an abatement of the symptoms. This form is common in the tropics, as it is most frequently malarial in origin. It may be due to other causes, however.

**INTERMITTING FEVER.** This type depends upon a fall to or below the normal, occurring usually once in the twenty-four hours. The paroxysms of fever run a definite course and are, as a rule, repeated periodically. This form occurs most frequently as a result of malarial infection or in pulmonary phthisis.

**RELAPSING FEVER.** This term is applied to a continued fever in which apparent recovery is followed by one or more relapses at intervals of about a week.

**INFLAMMATORY FEVER** is generally symptomatic of a local inflammatory process, and is out of place in a consideration of infectious fevers.



ADYNAMIC FORMS OF FEVER are attended by rapid loss of strength, a degree of failure which appears out of proportion to the rise in temperature; indeed, the pyrexia is not high. The pulse is small, feeble, and quick. The nervous system may or may not be disturbed, and the tongue is usually moist.

A condition present in various types of fever is described as the "*typhoid state*." It is often present in typhus and typhoid fevers, erysipelas, pneumonia, uræmia, and acute yellow atrophy of the liver. It is characterized by a dry, brown tongue, sordes upon the teeth and gums, feeble heart, weak, rapid, and perhaps arrhythmical pulse; feeble circulation in the superficial and pressure-bearing portions of the body resulting in inflammation and destructive changes, muttering delirium, twitchings, tremor, sinking to the foot of the bed; perhaps diarrhœa, which may be offensive, delirium, stupor, coma, and deafness.

HECTIC OR SUPPURATIVE FEVER. This form of fever attends upon a variety of affections, in which the prominent feature is protracted supuration. We meet it in pulmonary phthisis in its most typical character. The type is intermitting or remitting. The development is gradual, there being usually only a slight evening rise at first; this may increase to a remitting form of fever continuing for a long time, or remain intermitting in character developing into more or less complete paroxysms. The latter form often suggests malarial infection and leads to many diagnostic errors. The bright hectic flush over the malar bones, the wasting, and the clear mental state (even if the fever is long continued) are prominent symptoms.

**The Pathology of Fever** has received much attention during recent years. The process is apparently much more complex than has hitherto been supposed. The neurotic theory of fever is at present the one most generally accepted. There are few observers competent to express opinions who do not consider a disturbance of heat regulation as the first development in the morbid process. A study of the etiology of this disturbance indicates that the initial factor is of the nature of a morbid agent in the circulatory fluid, or that it may be an impression made upon the interested nerve-centres, either directly or indirectly, or reflexly. The origin of the blood irritant varies, and is as yet but partially understood. Alkaloids, whether the product of living micro-organisms or of metabolic processes (normal or abnormal), are the most important fever-producing agencies. Ptomaines may also be introduced from without, but of all external agents, micro-organisms, whether immediately pathogenic or acting as mediate agents, *i. e.*, exciting the necessary chemical changes for the production of a pathogenic agent, occupy the most important position. Aside from these, we meet a form known as thermic fever, due to exposure to heat; also a rise in temperature in various neuroses, and in organic affections of the nervous system.

The most important of recent contributions to the subject of fever are from the pens of Drs. MacLagan and Macalister. Macalister in his Gulstonian lectures on the "Mechanism of Fever," considers the nervous origin of fever in a rather original manner. He describes the thermal apparatus as consisting of three parts:

- (1) The thermotaxic or adjusting mechanism.
- (2) The thermogenic or producing mechanism.
- (3) The thermolytic or discharging mechanism.

The first relates to the stability of temperature; the second to heat production; and the third to heat loss. One or all of these functions may, according to Macalister, be disturbed in fever. Disorder of the first results in irregularity of temperature only; of the second with the first, in general elevation of temperature, increased body heat, ordinary fever; of all, in hyperpyrexia. It is pointed out that the development of these mechanisms is evolutionary, the cold-blooded animals being endowed with the mechanism that in mammals becomes thermolytic, *i. e.*, a nervous mechanism which governs the respiration and vessels. In young animals, the thermogenic mechanism is first developed. In infancy the special characteristic of the temperature is its instability, but with advancing years, thermotaxis is established. Macalister considers fever a "dissolution," *i. e.*, a relaxation of control from above downward. He cites recovery from typhoid fever as illustrating the hypothesis of "dissolution." The thermolytic mechanism is the first to be restored, indicated by a critical sweat or relaxed cutaneous vessels with resulting sudden loss of heat and consequent lowering of temperature, but until the thermogenic apparatus recovers, the temperature will vibrate for some days, and an epicritical excretion of uræa takes place with returning thermogenetic control, heat production is lessened and is counterbalanced by thermolysis. Thermotaxis is the last to be restored, for after the temperature is depressed to about the normal, it is marked by instability.

MacLagan, in his admirable clinical study of fever, calls attention to the physiological data to be considered in any study of the subject, and enumerates the component parts of the thermal apparatus as follows:

- (1) The tissues, especially the muscles in which heat is formed.
- (2) The surface from which heat is eliminated; about 80 per cent. escaping normally from the skin; less than 20 per cent. by the lungs.
- (3) A controlling centre in the nervous system.
- (4) Nerves associating this centre with the heat-developing structures.
- (5) Nerves connecting the heat centre with the heat-eliminating surface.

In the process of normal tissue metabolism, especially the kata-

bolism or retrograde metamorphosis of the tissues, certain products destined to elimination are formed, viz., urea, carbonic acid, and heat. Urea is eliminated by the kidneys, carbonic acid by the lungs, and heat by the body surface. In health a proper balance between production and elimination is maintained by the nervous system, the most beautiful illustration of which is seen in the preservation of the temperature of the body at an almost fixed point during the most diverse external conditions. In fever the amount of urea and carbonic acid is increased and the temperature rises above the normal point.

### ETIOLOGY OF THE INFECTIOUS DISEASES.

Each of the members of this group possesses a distinct individuality. They never merge the one into the other, although within certain limits variations in character appear. In reference to many, and by analogy, we are warranted in believing that all of the forms of disease represented by this class are due to the action of "pathogenic" micro-organisms. The special characteristics of the various organisms causing the forms of infectious disease are described in connection with the several articles treating of the same. In this section only the most general considerations relating to the germ theory of disease will be admitted. According to this theory the contagia are microscopic particles, probably of a vegetable nature, most of them members of the class called schizomycetes or fission fungi (Nägeli). Embraced by this division are the cocci, bacteria, leptotriches, and cladotriches. Many of these micro-organisms are widely diffused by the atmospheric air, and are also found in water and especially in the soil, as well as attached to innumerable objects in nature, and with the yeast and moulds conjointly produce the fermentation and putrefaction common to organic matter. The behavior of these microbes towards organic matter differs. Some subsist only upon living organic matter, while others attack both the living and the dead (pathogenic). Others attack non-living matter only, and are not concerned in the production of disease (saprophytes). The study of the pathogenic organisms is made difficult by our inability to cultivate all of them external to the human body. This is the case in respect to the micro-organisms of variola, measles, pertussis, etc. The germs of tetanus, erysipelas, diphtheria, anthrax, etc., possess a sufficient saprophytism to permit of their cultivation in artificial media, hence we possess much more definite information concerning them. Certain pathogenic organisms possess the quality of infection in high degree, pervading the fluids and tissues of the body in great number (septicæmia), while others develop at the port of entry and act upon distant parts through the media of poisonous alkaloids (diphtheria). Our ability to poison animals with sterilized cultures of pathogenic organisms with sufficient resulting lesions and symptoms to



identify the disease is ample evidence of the existence of such poisonous products. While micro-organisms of each variety multiply at a marvellous rate, if supplied with the necessary pabulum, their life history is usually very short, being destroyed in great numbers by opposing organisms, by their own products, or from a lack of proper food and environment. Preservation of the species is accomplished in some varieties through the development of spores. These appear within the germ as spherical (immature) or oval (complete) highly refractive bodies, which are finally freed through the destruction of the parent cell. It is seldom that more than one spore appears in a cell. These bodies resist ordinary stains, but may be colored by special processes, and they possess much greater resistance to injurious influences than do the cells from which they spring. Spores may retain life for long periods with the ability at any time and under favoring conditions to germinate into full-fledged micro-organisms. A study of sporulation has cleared up much of the supposed evidence of the *de novo* development of infectious diseases. In their propagation the bacterial species are true to themselves, as is the case with the higher and the highest species of plants and animals, modifications in character following only upon a changed environment. While the new characteristics are usually eliminated by a restoration to the normal environment, they may become permanent, and propagated with resulting development of new species. The modifications are usually of a retrograde character, and generally result from injurious influences. It has been demonstrated that the virulence of many pathogenic organisms may be lessened or eliminated by prolonged cultivation, by the action of many chemicals and by heat, light, drying, the blood-serum of some animals, extracts from the thymus gland, etc. The attenuated viruses have been employed much of late in therapeutics. Increased virulence has also been secured experimentally. It is a most interesting fact that certain of the pathogenic bacteria, especially the diplococcus lanceolatus, are found in the bodies of many healthy persons. Cultivation of such organisms and injections of the same into animals have demonstrated their true character. It is possible for a person to be affected by a double or mixed infection, *i. e.*, more than one germ producing its specific action upon the human organism at the same time, one may follow apparently upon the heels of the other. This is characterized as a "secondary infection." Streptococcus infections are common secondarily in diphtheria, tuberculosis, etc., and are responsible for many of the symptoms appearing in certain of the infectious diseases.

Recent observations indicate the more frequent co-existence of infectious diseases than we have in the past believed to be the case. Caiger, of Great Britain, recently communicated to the Epidemiological Society his experience at Stockwell during the past four years. During this period

he observed 362 cases in which two infectious diseases existed and seventeen with three diseases running some portion of their courses concurrently; in 200 of these the acute febrile stages of two or three coincided. Priority was calculated from the known incubation periods of the several affections. Scarlatina was the primary disease in 197. It was complicated by diphtheria, 97 times; varicella, 43; measles, 31; whooping-cough, 13; erysipelas, 10; enteric fever, 2, and typhus, in 1. Scarlatina existed as a complication in no less than 88 among 97, in which the primary disease was diphtheria; in 20 among 23 of varicella; in 14 of 17 of pertussis; in 6 of 9 of enteric fever, and 9 of 18 of measles, although in this instance diphtheria accounted for another 7. In the series of 17 triple attacks scarlatina was primary in 9, and a complication in 4 only, diphtheria holding the highest place with 9. During the past six years 48,367 cases of scarlatina were admitted to the hospital of the asylums board; of these 3,166 or 6.54 per cent. were complicated, 1,094 with diphtheria, 899 with varicella, 703 with measles, and 404 with whooping-cough; the relative numbers of diphtheria and whooping-cough being probably due to the fact that many had already had whooping-cough in infancy, while diphtheria, though less frequent, might and often did occur. These four diseases accounted for 3,100, or all but 66.

The questions suggested by these figures are: (1) Did any disease render the individual less or more susceptible to infection by another? (2) Did the primary disease affect the course or character of the secondary one in respect to (*a*) incubation, eruption, etc., periods; (*b*) severity of the disease; (*c*) distribution of local phenomena; (*d*) liability to sequelæ and other complications? Caiger's conclusions, after eliminating the influence of age, incidence, seasonal prevalence, actual frequency, etc., were unfavorable to the existence of antagonism between any of the infectious diseases, rather the reverse, there being both general and local increase in susceptibility; first, through lessened power of resistance: and, secondly, by the local inflammations favoring the growth of the contagia of diseases known to affect the mucous membranes or tissues in question. For instance, an attack of chicken-pox was without influence on any succeeding disease, but when scarlet fever was the primary disease, chicken-pox, favored also by the quasi-dermatitis left behind, might rival smallpox in intensity of fever and extent of the eruption. Diphtheria succeeding scarlet fever was grave in character, as the scarlatinal sore throat, rich in staphylococci and streptococci, presented a favorable soil for the Klebs-Loeffler bacillus. Also, while the general phenomena of measles might be aggravated by a preceding diphtheria or scarlet fever, and in the latter conjunction the rash would be intensified, diphtheria succeeding measles was more serious than the post-scarlatinal, since it inevitably attacked the larynx and trachea. Whoop-

ing-cough and measles were observed to co-exist, or to succeed one another, mutually increasing susceptibility. The incubation period of a disease was not observed to be much affected.

Micro-organisms enter the body from either the mucous or cutaneous surfaces. Some, such as the organism of tetanus, are more apt to attack wounded surfaces. The bacillus of tuberculosis enters most frequently by the respiratory tract; others, as the cholera germ, enter by the alimentary tract. While there are especially favorable locations for the entrance of the various pathogenic organisms into the human body, under favoring circumstances, the point of entrance may in some cases be by an unusual route, *e. g.*, inoculation of the surface of the body by the tubercle bacillus. It has been shown that the disease-producing influence of pathogenic organisms is, at least in many instances, in a direct ratio to the number of organisms introduced. It is believed also that the organisms vary much in their virulence, just as does the individual in his susceptibility. Those which possess the ability to propagate externally to the body by means of their greater numbers and wider diffusion, give rise to diseases of frequent occurrence. The most prominent points of location of the specific organisms vary much in different diseases. They are found in the stools, and sometimes in the vomited matters, in typhoid fever, cholera, etc.; in the sputum in pulmonary phthisis, pneumonia, etc. It is now believed that organisms can not be found in the breath unless ejected by coughing, sneezing, or other efforts, although the author's experiments reported in 1887 to the Pennsylvania State Society suggested the opposite respecting the tubercle bacillus. Elimination through the skin occurs in many of the highly infectious diseases, such as typhus. Many organisms are not thrown off as such, but are destroyed within the body. It seems probable that bacteria are never eliminated by organs in a healthy state, but that the tissues must first undergo damage in order to harbor them.

THE MEDIA OF CONVEYANCE OF SPECIFIC ORGANISMS from the infected to the healthy is not always the same. The most important are water, air, milk, ice, insects, and for the development and transmission of ptomaines, sausage, ice-cream, cheese, decomposing fish, etc. Perhaps the most widely known of these toxic ferments is the tyrotoxon of Vaughan, discovered by this pathologist. It is found in ice-cream, cheese and milk.

Much interest has recently attached to the question of the transmission of bacilli and their products from the mother to the fœtus, and it appears that the embryo may become infected through the mother or father (germinal infection). Syphilis and leprosy may be thus transmitted. This subject has received much attention respecting the transmission of tuberculosis. Baumgarten antagonizes the prevalent idea of heredity in that disease which contends only that a favorable soil for the



growth of the bacillus is transmitted by parents, rather than that the bacillus is transmitted to the offspring. He is strongly supported by recent experiments, especially those of Gärtner, who has demonstrated a maternal transmission of bacilli in canary birds. It seems probable that toxic alkaloids pass from mother to fœtus more readily than the bacteria that produce them.

*Water is one of the most important of the media of infection*,—pre-eminently so, as the spread of typhoid fever is largely due to water contamination by the bacillus of that disease. It is hardly less important in the more fatal and justly dreaded disease, Asiatic cholera, as well as in dysentery and certain diarrhœal affections. The life-history of pathogenic organisms in water has been carefully studied by many observers. Koch's observations upon the cholera bacillus are well known. They began some years ago during his trip to India, as a member of the Cholera Commission, and have been continued ever since, even during the recent epidemic in Germany. His observations, as well as much corroborative testimony, seem to furnish sufficient evidence of the specific element in Asiatic cholera being diffused in drinking water. The duration of life of the various species found in water differs much, and in the case of a single form depends upon many conditions. Koch found the cholera bacillus usually disappeared as early as the third day, but that its life may be extended to many times that period. The problem, as related to the typhoid bacillus, is a difficult one to solve, on account of the existence of a widespread bacillus resembling the pathogenic one. But in regard to this question *in toto*, it must be confessed that it is impossible to secure for experimental purposes the same conditions which exist in nature, and that knowledge gained from such experiments can therefore only approximate to the truth.

Temperature possesses an important influence upon the growth of germs in water as well as in other media; the higher temperatures favoring development, the lower inhibiting. Many bacteria are not destroyed by freezing, indeed many become active under favorable conditions after a residence of several months in ice. This suggests the danger which may lurk in this article when, as is often the case, it is procured from improper sources. Contamination of the water supply has been repeatedly demonstrated to exercise a predisposing influence, especially in typhoid fever and cholera. Whether this is due to an influence exerted upon the growth of these bacilli, greatly increasing their number or virulence, or whether due to the development of other micro-organisms, whose action upon the body favors the activity of the specific germs, is not entirely clear, but probably both, and other conditions as well are factors. In respect to typhoid fever at least, it has been shown that injections of a variety of micro-organisms or their poisonous products highly augment the susceptibility of certain animals to that disease. The various



ways by which drinking water becomes contaminated are now so well understood that pollution of this important fluid may be reduced to the minimum by proper effort on the part of sanitary authorities. But it also requires the earnest co-operation of medical men in general, who can be instrumental in teaching the people how to destroy infective secretions, and thus effectually prevent them from reaching the water supply. The presence of germs in large number in the upper strata of the soil has been referred to. It is assumed by Pettenkofer, Buhl and others, that when the sub-soil water is at a low level that these germs proliferate more actively in the dry surface soil, and are given more readily to the lower strata of the atmosphere.

*The spread of infectious disease*, especially of typhoid fever, *through the milk supply*, is now known to be quite frequent. Many milk epidemics of this disease have been reported. This important article of food is contaminated by means of the infected water used to wash the cans or dilute the milk. The great frequency of tuberculosis in cows, and the infection of their milk by the tubercle bacilli, has been recently demonstrated and indicates an important medium by means of which this disease spreads. Diphtheria, scarlatina and cholera may also be transmitted by milk.

The agency of *insect life*, especially flies, as carriers of infective organisms, has been positively demonstrated. One of the most interesting observations upon this subject was made by Sawtschenko, who fed flies upon cholera bacillus cultures, and found living cholera bacilli in their excrement three days later. The fact that micro-organisms become attached to various articles of clothing, furniture, domestic animals, etc. (*fomites*), and are by these means conveyed from the sick to the healthy, is well understood.

The *atmospheric air as a medium of transmission* for the spread of infectious disease occupies a much less prominent position in professional opinion of late years. While it is now certain that at one time far too great importance was ascribed to this means of diffusion of contagious principles, it is equally certain that at present it does not receive the attention it merits. This is another example of the old story of going to extremes. This change in opinion has been due to observation along many lines of research. The air which at one time was supposed to teem with disease-producing organisms has been demonstrated to contain but few. Even surgeons now operate without precautions for the destruction of germs in the air surrounding the wound. Other methods of infection have, moreover, been found to be more numerous than had been supposed. These facts account for many cases heretofore supposed to be due to infected air. The organisms of some affections, prominently malaria, are undoubtedly diffused freely by the atmosphere; others, such as syphilis, can hardly be supposed to be spread in this manner. The germs of many diseases (*tuberculosis*) are freely distributed in dust.

Dessication destroys some pathogenic organisms within a limited time. Others, such as the cholera bacillus, have been shown to live long enough to permit diffusion in dust (twenty-four to thirty-six hours), while the tubercle bacillus possesses great life-tenacity under these circumstances, permitting of cultivation after long drying. Recent observations as to the importance of dose in the development of infectious diseases suggest that the quantity of the agent taken in this manner may often be insufficient to develop an effect.

The specific germ is one factor in the production of infectious disease, the organism another. As all persons exposed to the same infection are not attacked by the poison, and as those attacked present variations in the intensity and character of the symptoms, we are forced to conclude that either the infective element varies in its disease-producing activity, or that individuals possess peculiarities rendering them unequally susceptible to its action. The observations suggesting this conclusion are numerous. If of a number of persons exposed to an infectious disease certain ones escape, we are apt to say they are not susceptible, but the conclusion is a most unreliable one, for, as Welch states, "it may be that the germs hit the one and miss the other." The truth of this is shown by the fact that those who at first escape may be attacked upon subsequent exposure.

That germs vary from time to time in their disease-producing power is shown by the variableness in type, intensity, symptoms, complications and mortality of infectious diseases in different epidemics. Susceptibility, or predisposition, may be general in character, concerning which we know little; or local, of which we have more exact information. Statements relating to general predisposition are not very tangible, and relate to impaired nutrition or tissue peculiarities, probably usually inherited, also all influences tending to lower nutritional activity, such as improper or insufficient food, imperfect sanitary conditions, overwork, mental or physical, or depressing mental emotions. Experiments upon animals have shown that susceptibility is increased by most of the above as well as by artificially produced anæmia, elevation of the temperature, overcrowding, fasting, etc. Local conditions favoring infectious disease are numerous. The protective influence of the gastric juice is well known, but it is partially lost in many disturbances of the stomach. Loss of muscular power in the organ and deterioration of the digestive fluid are most important changes favoring disease such as cholera, the infective agents passing this organ, which in health guards the lower tract, find a breeding-place in the intestines. Traumatic, inflammatory or destructive changes in the skin or mucous membranes open an avenue for the entrance of germs or their products to the tissues and fluids of the body. These injurious influences not only furnish a portal for the infective agent, but they depress the vitality of the part and lessen its

power of resistance. All conditions tending to local impairment of nutrition, and consequently of function, favor infection at the disturbed focus. General and local predisposing causes are frequently combined, as seen in phthisis.

*Age* is a most important factor. We need only cite the occurrence of scarlatina and a variety of so-called children's diseases in the young, and of typhoid fever from adolescence to mature or adult life. Outside of the sexual sphere, sex possesses little influence. Race shows some influence in some few of the infectious diseases.

### IMMUNITY.

Certain of the infectious diseases confer protracted or permanent freedom from subsequent attacks. This is especially true of the forms which are common to children. For instance, variola confers quite certain immunity from a second attack; and measles, pertussis, scarlatina, etc., scarcely less so. Many diseases afford only limited periods of immunity. Certain individuals, although frequently and thoroughly exposed to a most infectious disease, fail to contract it—they are insusceptible to it. The same observation has been made regarding exposure of certain of the lower animals to the infectious diseases. It is probable that while in general a period of diminished susceptibility follows upon an attack of any of the members of this group, this is not always the case. Indeed, the reverse may obtain, and an increased susceptibility be awakened, as evidenced by the occurrence of a second attack within a very short time. This is especially true of influenza, typhoid fever, pneumonia, etc.

Extended experimentations, having in view the artificial establishment of immunity from various infectious diseases, have been conducted during recent years. The first efforts in this line were made by Lady Montague in 1717, who advocated inoculations with the virus of variola. In the middle of the eighteenth century vaccination with the virus of kine-pox was introduced to secure immunity from smallpox. It was first performed by Benjamin Jesty, an English farmer, and later firmly established as a prophylactic method by Edward Jenner. These researches have progressed actively during the past decade, or since Pasteur, in 1880, discovered a successful method of vaccinating for chicken cholera; and since that date there is hardly a disease of known or suspected bacterial origin which has not been subjected to vaccination experiments, and in many instances periods of immunity, or some degree of immunity, have been secured. Extensive consideration of this subject is not possible within the scope of the present section; but it is a subject so full of interest, and one which has thrown so many side lights upon medicine, that it merits careful consideration. The inoculation methods employed have been various. (1) Inoculation of



persons to be rendered immune with cultures of the specific germs of the disease, either in their virulent condition or variously attenuated. (2) Inoculation with the products of the activities of pathogenic organisms; these substances being employed while still toxic, or partially deprived of their toxicity by means of admixture with chemical substances, the action of heat, or combination with animal juices. The discovery that this class of non-living substances possesses the power of granting immunity has been a decided and important advance in our knowledge. Another fact of interest is the greater activity of the non-toxic cultures and filtrates in the production of immunity, which suggests that the toxic and immunizing products are independent of each other. (3) The fluids of immune animals, especially blood-serum and milk, have been employed, both as preventive and curative agencies. Experiments looking to the production of immunity through the use of bacteria other than those specific to the disease, have been performed with indifferent results; but it is claimed that immunity from a specific disease may be secured by a variety of agents, or by a combination of agencies.

The introduction of these vaccines into the body is followed by reactionary symptoms of varying intensity, immunity succeeding upon their disappearance. The duration of the period of reaction varies from a few hours to days. Even periods of fifteen or twenty days have been reported, the duration depending upon the character as well as the dose of the poison. When living micro-organisms are introduced they multiply at the point of inoculation and attack the organism through the agency of the poisonous substances which they elaborate. They may also invade the fluids and tissues of the body; but this is not essential to their efficient action. A certain amount of local inflammation and pyrexia are common attendants; indeed, an attack of mild severity of the disease represented by the germ employed may result. The chemical products of bacterial life appear less likely than micro-organisms to occasion local inflammatory changes.

Experiments seem to demonstrate that in the course of certain infectious diseases in human beings substances are produced which possess the property of rendering animals insusceptible to the influence of the germs of these diseases. Lazarus found the blood-serum of persons convalescent from Asiatic cholera sufficient to create immunity for that disease in guinea-pigs; and this property of the blood-serum was demonstrated by Wassermann to exist as late as forty days after recovery. Normal blood-serum has been shown to possess in some degree the same power. It is supposed that an immune person or animal possesses in the blood substances which destroy the infective agent when received. Such substances are called antitoxins. The manner of action of an antitoxin has not yet been determined. In

this relation the most important observations have been made upon diphtheria and tetanus. A supposed antitoxic power of the blood does not, however, explain certain immunities; for instance, hereditary immunity from tetanus, or immunity from certain affections when the blood is teeming with bacteria, viz., anthrax. Under these conditions the tissue-cells possess a not understood tolerance of or resistance to the specific poison. For the discovery of immunity through the action of milk, taken as food or injected, we are indebted to Ehrlich. It suggests interesting thoughts in respect to the influence of hereditary tolerance. Regarding immunity gained by transmission from parents much has been written, but little is definitely known.

Whether we ascribe immunity to a condition of the fluids of the body or to a quality of the tissue-cells, matters little at present, as the subject is not more than in its infancy; and the one is closely related to the other, the fluids being the product of the activities of the cells.

A novel and plausible theory regarding immunity has been advanced by Metschnikoff, and is known as the theory of *Phagocytosis*. His first paper appeared in Virchow's *Archives*, 1884, entitled "Blastomycetic Disease in Daphnia—a Contribution to our Knowledge of the War of the Phagocytes against Pathogenic Microbes." This has been followed by a number of papers of a most interesting character by the same observer. Of special interest is the one upon "The Relation of Phagocytes to the Bacillus anthracis." He states that if the bacillus anthracis is injected into the tissues of the frog, the leucocytes may be seen to attack the bacilli, incorporating and then destroying them. Under certain conditions, however, such as subjecting the animals to a high temperature after inoculation, the bacteria propagate rapidly, gain the ascendancy, and the animal becomes ill and succumbs to the infection. The same process has been observed in the study of pathogenic organisms in the blood of animals. This view places the leucocyte in the vanguard as a defender of the organism against the invading disease-producing micro-organism. This protector is designated a *phagocyte*. While the white blood cell is the most important phagocyte, the same function is performed by other cells of mesodermic origin. According to this theory, immunity is gained when tolerance of a bacterial poison exists, the phagocytes being no longer overcome by the invading organisms. Fascinating as this theory appears, it is yet to be demonstrated, however, that the injury to the micro-organisms may not be produced by the fluids before they come into contact with the fighting cells, thus rendering them easy prey. The known injurious influence of the fluids of the body upon pathogenic organisms can hardly be ignored in the construction of this or any theory of immunity. Metschnikoff does not accept the observations of many investigators which indicate that the fluids of the body possess the power of destroying

disease germs, and that many of these die and disintegrate external to the cells. Attractive as this theory appears, it can be considered as telling but a portion of the truth relating to immunity.

To even notice the various doctrines of immunity would consume too much space, but it may be said that the most prominent are, first, one which teaches that during an attack of infectious disease a chemical substance is elaborated which, continuing in the body, resists future attacks of the organism. Secondly, that during an attack the micro-organisms consume all available pabulum for their nutrition, leaving an exhausted soil insufficient for future propagation of the species. Thirdly, the doctrine of natural immunity, which asserts that the tissues of certain persons or animals possess a vital quality of resistance to the development of certain microbes.

### METHOD OF EXAMINATION OF INFECTIOUS DISEASE.

As the care of cases of infectious disease constitutes a considerable proportion of the daily occupation of the general practitioner, it is important that he should establish exact methods of procedure in their investigation, for it is only thus an early diagnosis can be made, a thing of the greatest importance from both a prophylactic and therapeutic standpoint. It is best to first inquire after possible causes of the present sickness, and especially as to whether the patient has been exposed to any disease of an infectious nature. It is also well to learn what diseases of this character the patient may have previously suffered from. The endemic or epidemic prevalence of any disease, and any state of the patient's environment which might possess an influence upon the question of contagion, must not be overlooked. Secondly, the mode of invasion must be carefully studied. This is often neglected. If possible, the day, even the portion of the day, upon which the symptoms appeared, should be determined. If this is neglected it is often impossible to recall the facts later. It should also be determined if the patient has had a failing condition of health prior to the onset, *i. e.*, premonitory symptoms. Such symptoms are quite common during the few days or weeks preceding many infectious diseases. My observations have convinced me that there is considerable of sameness in the character of these precursory symptoms as observed in association with different affections. They consist of varying degrees of pain in the head, back and limbs, malaise, anorexia, and numerous symptoms associated with the digestive tract, vertigo, mental depression, etc. If the attack of fever is due to a local affection, such local disease will be generally indicated by pain and other symptoms in the affected region. It must not be forgotten, however, that infectious diseases are frequently complicated by a variety of local symptoms, which, if occurring early, are very likely to lead astray. I have just witnessed a case of typhoid fever in which the most annoying symp-



tom was a most intense pain in the right hypochondriac region, continuing for days. It is not uncommon to meet with such a prominence of pulmonary symptoms in the same affection as to mislead for a time the most skilful observers. The danger of error in diagnosis is in the early stage, when it is most important that mistakes should not occur. A study of the course of the fever is essential, and of great assistance in early diagnosis. This is accomplished by the thermometer, the manner of using which instrument is described in another portion of this section. The various fevers have typical temperature curves, modified quite frequently by the occurrence of complications, and more or less by the epidemic, treatment, etc. The temperature range is also of prognostic importance. In order to a just estimate of the condition of a patient suffering from infectious disease a systematic examination should be made from one to three times in the twenty-four hours, the frequency being determined by the severity of the symptoms. In these days of trained nurses, clinical thermometers, and other instruments of precision, there is danger of neglect of certain methods of examination, especially those which involve the use of the special senses. This is unfortunate, as a most comprehensive investigation is essential to a full understanding of the case. A frequent error is the too great study of the disease, and the too little study of the individual. It may be stated as an axiom that *one of the first duties of the physician in his attendance upon a febrile case is to discover the physical condition of the patient, i. e., by means of physical exploration and chemical analysis to learn the degree of integrity of the heart, lungs, kidneys, and digestive tract.* Nor should the nervous system escape attention. The heart and kidneys may be mentioned as the most important, for it is these which "bear the heat and burden of the day," in protracted febrile affections. The conditions which underlie an unfavorable prognosis may be often discovered as well upon the first visit to the patient suffering from typhoid fever, as late in the second or third week, such conditions being usually discovered by means of physical examination of the chest or urinary analysis, methods of examination which should be made frequently during the course of any infectious disease. For instance, the early supervention of pulmonary or cerebral symptoms or of gastric irritability is often due to a kidney lesion which has as a rule been overlooked. And it is equally true that arterio-sclerosis, and a variety of heart affections, are responsible for the early development of serious symptoms.

A skin eruption is a marked feature of most infectious diseases. Each eruption is also characteristic of the disease with which it is associated. Beside the typical rash, other forms may often be observed, initiating, coexisting with, or concluding the eruptive period. It must be remembered, however, that in the continued fevers and even in the exanthemata, the eruption may be absent; also when infection has



occurred by inoculation the eruption may be confined to the region of entrance of the poison. The facts which should be determined, if possible with reference to an eruption, are its character and the time of its appearance as related to the onset of the attack, especially the fever. Of scarcely less import is a determination of the region of the body upon which it first appears, and the manner of its development and extension. It is also well to learn as to the order of development of the eruption in other portions of the body. The duration and the sequential conditions, such as desquamation, etc., are important.

**THE CLINICAL THERMOMETER AND ITS USE.** This invaluable little instrument is in this day too well known to make a description necessary. It can be procured from any surgical instrument dealer of reliable character at small expense. The best makers accompany their instruments with a certificate stating the amount of variation from a recognized standard. As thermometers change with age and use, registering too high after about a year, it is necessary that they should be occasionally compared with such a standard. This is done by the maker at a small cost. A plan of my own has been the comparison of instruments for correction with a new thermometer of the best manufacture. This is accomplished by placing the two thermometers in water of about the temperature of the lowest marking upon the scale, and raising the temperature gradually to the highest, noting the variations at every five degrees. A record should be made of these. An opportunity for correction occurs whenever a friend buys a new instrument. Very delicate methods of determining temperature, as by means of the thermo-electric apparatus, are never used by the practical physician, and need not be described. Instruments for the taking of surface temperatures are constructed with an expanded mercurial bulb at right angles to the stem. Only self-registering thermometers should be purchased.

**APPLICATION OF THE THERMOMETER.** The temperature of the body may be taken in the mouth, axilla, rectum or vagina. For reasons of delicacy the first is employed, unless more accurate observation is demanded. The rectum and vagina give the highest registrations, the mouth somewhat less, and the axilla nearly one degree less than the rectum or vagina.

*The buccal temperature.* In securing mouth temperatures the bulb of the thermometer must be placed beneath the tongue, and the lips, not the teeth, closed firmly about the instrument. In children the bulb is liable to be broken by the teeth. Breathing must be carried on entirely by the nostrils. Four or five minutes will give a correct rendering. The taking of ice or cold liquids prior to the observation may affect its accuracy.

*Axillary temperatures.* If moist, the axilla should be carefully dried. In placing the instrument it is important to see that it is inserted high in the armpit, and is kept well anterior to the posterior fold. The arm

should be held close to the side or drawn across the chest. Five to seven minutes are necessary to secure a correct registration.

*Rectal observations* are most reliable, and should always be taken in case doubt exists as to the true temperature. Errors may result from the presence of feces, the bulb of the thermometer being thrust into the mass. To avoid this error the rectum should be first emptied by means of warm water. The instrument should then be oiled and the bulb buried well within the sphincter muscle. Two to four minutes are sufficient for a correct rendering. This method is very convenient in young children, for use during the bath, and in all cases in which the surface circulation is impaired. Other methods are sometimes employed. In children especially the bulb of the instrument may be enveloped in the folds of the groin, or between the tissues of the groin and the flexed thigh. If the urine as it flows from the urethra is allowed to fall upon the thermometer bulb, it will give the exact temperature. This method has been satisfactorily employed in the detection of malingering.

The frequency with which the temperature is registered will depend upon the nature of the case. General practitioners seldom take the temperature oftener than once in the twenty-four hours unless more than one visit is made, or a nurse is in attendance; and as the usual hour of visit is not the period of maximum or minimum temperature, it is a most unsatisfactory method. There should always be a morning taking (minimum temperature), also one in the late afternoon or evening, the time depending upon the time of apparently highest temperature (maximum temperature), but in many cases it is important that frequent observations should be made. The taking of the temperature should not be stopped with improvement, as the first evidence of an aggravation of symptoms may be a return of the pyrexia. The pulse and respirations should be taken and registered at the time of each taking of the temperature. The information afforded by the thermometer is of such importance in diagnosis and prognosis, and furnishes such positive indications for therapeutic action, that it is deemed best to consider shortly its relationship to these subjects.

The keeping of systematic temperature charts is a very important matter, but one, nevertheless, which we all neglect far too frequently. One is enabled by a single glance at a properly prepared chart to note the progress of the case from its inception. Then again these charts, if properly filed, afford a source of scientific information that cannot be underestimated. Most of the charts in use provide for morning and evening temperatures only, with blank spaces for insertion of the pulse and temperature respiration rates. Bashore's chart has provision for temperature, pulse and respiration curves; that in use in the Hahnemann Hospital has spaces for four observations daily, and a separate chart for the pulse curve. Any of these charts may be had at small expense.

**DIAGNOSIS.** The thermometer not only enables us to determine the presence of fever, but to learn of its degree and course, features which frequently permit us to make an early diagnosis. All are acquainted with the characteristic temperature range of the early days of typhoid fever, which is sufficiently often present to render it of value in diagnosis, days before the typical eruption has appeared. Even the persistence of a moderate rise for several days, if unaccompanied by apparent cause, always excites in my mind a suspicion of typhoid fever, as the majority of cases of continued fever met in this region, not due to pulmonary phthisis, are typhoid in character. A rapid rise within a few hours, a correspondingly rapid fall of temperature, if occurring in one who has been in ordinary health, is strongly suggestive of malarial infection. Taken in association with the symptoms and history of the case, the rapidity of the temperature rise is of great diagnostic significance. However much the symptoms may suggest scarlet fever, for instance, the absence of the rapid temperature rise of that disease will enable the observer to throw strong doubts upon the probability of the correctness of the supposition. The sensitive character of the nervous and circulatory systems in children must be remembered, or undue importance will often be attributed to the sudden rises of temperature met in these subjects. These illustrations, which might be increased, indicate the importance of a thorough study of the temperature range of the various infectious diseases, especially in their early days. The presence of an abnormal temperature frequently leads to the discovery of unsuspected disease. A regular evening rise as well as a subnormal morning temperature, have often excited suspicion of the existence of pulmonary disease, which suspicion has been proven well founded by recourse to physical diagnosis. The occurrence of abnormality in the temperature range not uncommonly gives warning of a complication. In typhoid fever a sudden fall of temperature may occur in connection with a hæmorrhage into the intestine which has not yet appeared externally, or again it may occur in association with perforation of the bowel. The temperature range is also suggestive of the variety of a certain disease which may be present, viz., phthisis, and is also directly related to the intensity of the morbid process. The thermometer also gives valuable information as to the development of inflammatory action subsequently to hæmorrhage into the various organs, especially the brain and lungs. In some diseases the superficial temperature may be much lower than that of the deeper parts. This may be often observed in cholera and other affections in which there is rapid loss of fluids, or failure in the circulation. Localized abnormality in temperature is a phenomenon of frequent occurrence in lesions of the nervous system and is the result of vaso-motor disturbance. The example most frequently met is the elevation or depression of temperature so often observed in paralyzed parts. Also in



neuralgia, hysteria, various lesions of the brain and cord, local changes in temperature are observed.

PROGNOSIS. The importance of the temperature in prognosis is even greater than in diagnosis. Much can be judged of the probable severity of a developing disease by a study of the initial temperature range. Excluding the early high temperatures occasionally observed in young excitable subjects, which usually soon subside, it may be stated that an early and rapid rise to a high point is indicative of a severe attack, regardless of the nature of the disease. The same statement also applies to the later periods of febrile affections. The significance of high temperatures is greatly increased if associated with defective excretory processes. It is in these cases that the group of symptoms designated "typhoid" frequently appears. The height of the temperature at certain periods of a disease is of prognostic importance. For instance, Loomis states that "the height of the temperature upon the eighth day determines the range of temperature that may be expected upon each succeeding day" in typhoid fever. Abnormal fluctuations of the temperature in the course of any febrile affection which pursues a typical course is significant of some complication or irregularity and indicates the necessity for thorough investigation. The presence of a low temperature while the general symptoms are of a serious nature, or a marked fall in temperature without a corresponding mitigation in the general symptoms, are both of unfavorable import. Very low temperatures ( $95^{\circ}$ – $93^{\circ}$ ) are always of ill omen. The influence of the thermometer in *therapeutics* is also considerable and worthy of mention. With it we often discover the presence of hyperpyrexia before the symptoms have suggested its presence, and are thus able to apply earlier more energetic treatment in suitable cases. By means of the thermometer is the employment of cold water in the treatment of fever regulated. It adds much to our knowledge of convalescence, being an important factor in determining the proper time for granting solid food and permitting exercise. It gives early warning of relapses, enabling the physician to apply treatment quickly. It often gives evidence of the non-agreement of certain food and thus assists in the selection of a proper dietary.

The first duty of the physician after determining the infectious nature of a patient's ailment is to adopt measures for the prevention of its spread. These will differ somewhat according to the seriousness of the disease. The following rules represent the best practice of the profession in this matter.

(1) *Immediate isolation of the patient*, preferably upon the upper floor of the house, should be insisted upon. If convenient, this floor should be reserved for the patient and his attendants. The attendants should associate as little as possible with others, and then only after change of

clothing and a walk in the open air. Attendants should wear in the sick-room only clothing to which micro-organisms do not readily adhere. Nurses should keep their finger-nails short and clean their hands carefully with the nail-brush, following this process by immersion of the hands in a disinfectant solution. If the nurse is a male he should have a clean-shaven face, and keep his hair closely cropped, which admits of more perfect cleansing. Physicians should make short visits and take proper precautions to avoid acting as fomites.

(2) *Preparation of the room.* The room should be large, well ventilated and light, but with arrangements for shading if required. All useless furniture, especially the carpet and all upholstered articles, curtains, etc., should be removed before the patient enters. Mats which may be burned are allowable. An open fireplace is desirable, and if the weather permits, a fire should be kept burning. It is the author's opinion that the time has arrived when every residence of sufficient size should be constructed with a hospital-room upon its upper floor, answering to the above requirements. Until such a step makes it easier to isolate the infected, it seems probable that infectious disease will flourish in our midst. Visitors should be prohibited, even members of the family, who are not essential to the sufferer's welfare.

(3) The strictest cleanliness of the patient, the room, and of the attendants, should be insisted upon. It is extremely rare that the condition of the patient will not permit daily sponging of the entire surface of the body; this is a greatly neglected duty and one which physicians are quite prone to overlook, perhaps for the reason that it is assumed that the nurse will attend to it without instructions. I desire to enter a protest against allowing patients to go for days, and even for weeks, without the use of warm water and soap, which is a common practice with many. It does not seem necessary to advance arguments in support of this practice. The bedclothing should be changed daily, the floor frequently cleaned and sprinkled with an odorless and efficient disinfectant. The door should be screened with a sheet kept moist with a disinfecting fluid. Some hang such a screen upon both sides of the door. All discharges should be received in vessels containing a germicidal solution, and allowed to remain long enough to be thoroughly acted upon. Closets and washstands should have disinfecting fluid poured into them daily. The impregnation of the atmosphere with offensive disinfectants is an annoying and useless procedure. All articles of clothing and bedding, also all dishes or other things of whatever character, must be subjected to similar treatment before they are taken from the room. Before leaving the hospital-room the patient and attendants should bathe, shampoo the hair and apply proper disinfecting solutions to the surface of the body. After this has been accomplished fresh clothing is to be brought, and after donning the same they are to immediately leave the room.

(4) After vacating, the room should be promptly cleaned and disinfected. It is necessary in this connection to call attention to the fact that developing a bad odor in a room does not mean its disinfection. The only efficient method is the thorough cleaning of every square inch of the apartment occupied, by means of soap, water, brush and disinfecting fluids applied directly to the entire interior of the room. If the walls are covered with paper it should be removed and whitewash applied; also a fresh coat of paint should be applied to the woodwork. According to many authors these measures are to be more or less thoroughly employed according to the degree of contagiousness of the disease. But this is a wrong principle of action. If we desire, as we should as good citizens, to diminish the prevalence of contagious diseases of all character, both mild and severe, the same rules of action should guide us in our treatment of all forms. It is highly desirable in the case of many infectious diseases to determine the sanitary condition of the premises, as many such affections are due to defective plumbing, or other causes of unsanitary conditions. Such inspection frequently results in the discovery of the origin of an infectious disease which has attacked one after the other of a family continuing the disease in the house for a period of time.

PREVENTION AND CONTROL OF EPIDEMICS. When an epidemic of an infectious disease is imminent or already present in a community, it becomes the duty of the physician to instruct the laity as to the proper means of its prevention and control. In large cities this information emanates from boards of health, but in the smaller towns and hamlets the general physicians must be looked to for the necessary knowledge. Physicians practising in such localities should therefore make themselves familiar with the subject, and even in cities much depends upon the physicians, who come into immediate touch with the people. Cleanliness of person and of habitation are of first importance. The former needs no comment. As to the habitation, it is necessary to remove all decomposing organic matter which may be found in or about the premises. Cellars especially should be thoroughly cleaned, disinfected, and whitewashed. Everything associated with the drainage of the house should be inspected by a competent individual. Nothing should be taken for granted. The water supply should be carefully investigated. The frequency with which the drinking water acts as a medium of transmission of the specific germs of typhoid fever, cholera, and other infectious diseases, is well known. The propagation of pathogenic organisms in water, at all freely, requires above all else contamination with organic matter. Drinking water thus fouled will, particularly during the warmer months of the year, furnish an excellent cultivation medium for certain of these organisms, which are responsible for a large percentage of our infectious fevers. Experience during the recent cholera epidemic at Hamburg has strongly corroborated previous observations. The water



supply in most cities is derived from rivers which drain large tracts of country, and which contain constantly disease-producing organisms, especially the bacillus of typhoid fever, the active development of which is largely dependent upon organic impurities. Wells of drinking water may be fouled in a variety of ways, frequently by receiving drainage from a neighboring privy vault. The water supply may also become contaminated within the house by means of leaky drainage pipes opening into the water-pipes. The source of the milk supply should be investigated, as contaminated water is often used to dilute the milk, or to wash the cans. It is best to boil all milk and water during epidemic periods, indeed it is a wise precaution at all times, if the drinking water is open to the slightest suspicion. Next in importance to the prevention of infection is the preservation of the most healthful possible condition of those who are exposed. This is best accomplished by the best of habits in reference to eating, drinking, exercise, sleep, etc. Those who attend upon the sick should observe these precautions with especial care. They should never allow the stomach to become empty, or themselves very tired while in attendance, neither should they neglect sleep nor daily exercise in the open air.

If persons suffering from infectious disease cannot be properly cared for in their own homes they should be removed to a hospital for contagious disease. It is also best if the outbreak occurs in a densely populated region, or in a crowded tenement house, to remove the patient as quickly as possible, and take every precaution for the prevention of the spread of the disease. If an ambulance cannot be procured the vehicle employed for transporting the patient should be thoroughly cleaned and disinfected. A conveyance which is not upholstered should be employed. The use of public conveyances for this purpose is criminal. During the progress of an epidemic the physician should accept no more cases than he can properly attend to, and no more than he can care for and preserve his physical condition, for only so can he do justice to those who intrust themselves to his care.

**Treatment of Fever.**—A most cursory review of the therapeutics of fever teaches one that diverse views are still entertained regarding many features of its treatment. This is especially true of the dietetics of fever.

**Food.** There have been two well-defined eras in the food-history of fevers. The first one, characterized by under-feeding, terminated with the illustrious Graves. With him began the development of the present plan of over-feeding, which it is true is waning; with the recent advocacy, by a number of observers of prominence, of a fasting method, especially when combined with complete rest. I have for years antagonized the prevalent stuffing plan, considering that very moderate quantities of food are sufficient, even for subjects of the protracted infec-

tious fevers. This practice is founded upon both reasoning and experience. Inflexible rules cannot be laid down for the selection of food for a fever case. Cases must be individualized. It is certain, however, that in the majority a strictly liquid diet is best tolerated. Of liquid food, milk is the most valuable. Animal teas, eggs and long-cooked farinaceous foods are advocated by some, but if good milk can be procured, taken and be well digested by the patient, it is usually preferable. Whatever the food selected, it is important that it should be administered at frequent regular intervals and in definite quantities. There is altogether too much laxness in respect to this feature of diet in fevers. The same may be said in reference to the resumption of general diet during convalescence, the tendency being to the too hasty return to solid food and a varied dietary. Marked prostration is by most counted as an indication for the giving of large quantities of food, which in my judgment is a grievous error, it rather suggests careful selection, preparation, and administration of food. The amount of food which the organism is capable of appropriating under these circumstances is but small, and a large quantity acts as an irritant. Water is as important as food, yet how seldom is it systematically prescribed, *i. e.*, in a given quantity in twenty-four hours. Distilled water is preferable; as it favors a freer excretion of solids. Larger quantities may be introduced by making use of the rectum several times in the twenty-four hours. If typhoid symptoms are threatening in their severity, and satisfactory amounts of water cannot be introduced, it should be administered hypodermatically. This is easily accomplished by the aid of a fountain syringe and a perforated needle. Boiled, distilled water, containing one-tenth of one per cent. of chloride of sodium should be used for this purpose. The loose tissues of the abdominal walls present the most favorable location for the operation.

STIMULATION by means of alcoholics is a very general practice. Indeed it is so much of a routine plan of treatment that many practitioners prescribe stimulants for nearly all cases of protracted fever, regardless of indications. Such indiscriminate use of alcoholics is productive of much harm. While a stimulation plan of treatment is in many cases of great value, my personal estimate of it has rather waned during recent years. My own cases reported to the American Institute of Homeopathy in 1888, consisting of two series of 100 cases each, with the lowest mortality reported, for unselected cases, *viz.*,  $2\frac{1}{2}$  per cent., were treated almost without the aid of alcoholics. Good old whiskey or brandy is the most to be recommended; wine is much used by some, and beer by others. Strong alcoholics should always be diluted, and unless given for urgent symptoms, should be administered in definite quantities and at stated intervals. The indications for the administration of stimulants are, first, progressive enfeeblement of the

heart. This is judged of by the character of the heart's impulse, and by the sounds of the heart; also by the force and frequency of the pulse, and the state of the superficial circulation. Equally suggestive of a stimulant plan of treatment is a high grade of delirium, pulmonary consolidation or œdema. If the action of a stimulant is favorable it is manifested by lowered temperature and lessened delirium, by sleep, a moist tongue, by lessened frequency of the heart's action, and of the respiration, also by an increase in the tone of the pulse. The opinion was at one time entertained that alcohol possessed an important influence in the reduction of febrile temperatures, but experience has demonstrated that the amount of reduction that can be secured by this means is, as a rule, too little to be of material value, and even then requires the administration of objectionable quantities. It should be emphatically taught that *stimulants are to be administered only upon the presence of well-defined indications, and that their action should be watched with as much care as we bestow upon the action of any medicine.*

TREATMENT OF SPECIAL SYMPTOMS ASSOCIATED WITH FEVER. Careful attention should be given to the excretions. This is carried to an extreme by certain practitioners, who, by means of their active eliminatory methods, frequently do much harm. At the other extreme are those who show an utter want of regard for the subject. The bowels are most frequently neglected, resulting in the accumulation of scybalous masses which irritate the intestinal mucous membrane, sometimes even exciting ulceration, and furnishing poisonous matters for absorption. This is most frequently the case in typhoid fever.

It is good practice to move the bowels every twenty-four to forty-eight hours by means of an enema. If this is unsatisfactory a simple laxative may be administered. Laxative medicine is not admissible in typhoid fever, at least exceptions to this rule are rare. In certain diseases, especially typhoid fever, diarrhœa is considered by many an advantage if kept within moderate bounds (two or three stools in twenty-four hours). The urinary secretion is usually decreased in its watery element, but contains an increased amount of solids. The latter are apt to be diminished with the progress of the febrile attack. This is due to a variety of conditions. For instance, degenerative changes in the renal epithelium impair its functional activity, lessening its excretory power. The character of the diet furnished to fever patients is also in great measure responsible for the small amount of urinary solids. Before directing treatment to the urinary secretion, the bladder should be examined and if there is a suspicion developed that the urine is retained, the catheter should be passed, making use of antiseptic precautions. The free use of water, especially distilled water, favors the excretion of urinary solids. Medicinal diuretics are rarely called for or useful. As there is usually an excess of nitrogenous waste matters it is generally best to use nitro-



genous food with great moderation. The skin should receive careful attention. Frequent cleansings with warm water and soap and frequent spongings with warm or cold water, general baths or wet packs, as indicated, exert a powerful influence upon the cutaneous excretion. Diaphoretics are rarely required. If indicated, the least disturbing to the patient and the least objectionable on account of non-interference with other treatment is the dry hot-air bath. This is applied by means of a tin funnel, shaped like an ear-trumpet. With the patient and the smaller extremity of the instrument between blankets and a lighted alcohol lamp beneath the larger funnel-shaped end, a current of dry hot air is caused to circulate about the patient. Another method is to seat the patient with an alcohol lamp beneath the chair, both patient and chair being thoroughly enveloped with blankets, or in the same position steam may be introduced beneath the blankets.

**GASTRIC SYMPTOMS.** Thirst is a symptom which is almost constantly present in febrile affections, and it is wise that this is so, for fluids in large quantity exercise an excellent influence upon the poisoned blood and tissues and favor the excretion of morbid products. Even if thirst is not present these patients should, for the above reasons, be encouraged to drink; the best of all drinks is water, which may be iced, carbonized, or in some cases impregnated with well-cooked barley, or rice, toast, and various fruit juices, such as lemons or tamarinds, apples, oranges, etc. Nausea and vomiting are not uncommon, especially in the early stage of the eruptive fevers, but usually quickly subside without treatment. If it can be positively determined that the patient has partaken of irritating matters the stomach should be emptied by means of an emetic. The evidence upon which many practitioners administer emetics is, however, unsatisfactory. For the control of troublesome vomiting it is necessary to put the patient upon a very limited diet or to grant no food whatever by the stomach. The taking of very hot water, carbonized water, champagne, small quantities of iced brandy, or the swallowing of small pieces of ice after they have been smoothed by the heat of the mouth, all sometimes exercise a favorable influence. Hot, cold or irritating applications to the epigastrium are useful adjuvants. It is best to keep the patient as quiet as possible, to avoid mental excitement and all confusion, in a cool, well-ventilated room. Various medicines, such as *ipecac*, *creasote*, *arseniate of copper*, *apomorphia*, various salts of *antimony* and *veratrum album*, prove of great service.

**NERVOUS SYMPTOMS.** The symptom of this class which usually calls for special consideration or treatment is headache. If the disease is a protracted fever it is best to cut the hair short at once, a procedure which usually brings a marked degree of comfort to the patient and permits a satisfactory application of hot or cold applications. This is of little consequence if the fever is of a protracted nature, as the hair is liable

to be lost. Ice bladders enveloping the cranium, or applied to the nape of the neck, are most frequently beneficial. To secure the best results they should be employed early and continuously. The cold applications not only relieve headache, delirium, and sleeplessness, but favor reduction of temperature. Small dry cups to the temples, forehead and nape of the neck sometimes afford valuable help. Elevation of the head and shoulders is sometimes beneficial. Of course our reliance is mainly upon medicines of which *belladonna*, *gelsemium*, and *cuprum aceticum* are the most important.

**DELIRIUM.** Delirium often constitutes the most troublesome symptoms of a fever case. Much may be accomplished by means of good general care in the direction of prevention as well as modification if the symptom has become established. A cool room, a clean skin sponged frequently with cool water, cold applications to the head, and proper attention to the secretions will often exercise a marked influence. Stimulants are indicated if the case is of an adynamic character. For the milder forms of delirium it is not necessary to select a special medicine, the remedy which best corresponds to the totality of the symptoms proving sufficient. For the higher grades, the most useful remedies are *belladonna*, *hyoscyamus*, *opium*, *stramonium*, or the most recent remedies *agaricine*, and *hyoscine*.

**SLEEPLESSNESS.** Sleeplessness is a symptom which too frequently receives an insufficient amount of attention, its neglect frequently results in an irreparable prostration which may determine a fatal issue. The measures already suggested for the control of headache, delirium, etc., are in the main indicated for the sleepless patient. With a proper use of these measures and the careful selection of general remedies it is rare indeed that we have to resort to opiates or any of the well-known hypnotics.

**RESTLESSNESS.** This symptom may be associated with other nervous phenomena or stand quite alone. If associated with delirium, etc., it does not require special treatment, being controlled by the means adopted for the control of coexisting symptoms. When not associated with high-grade nervous symptoms it is remarkably within the control of *rhus toxicodendron*. Cold packs to the head, spine or abdomen are useful. Rarely, hot applications may prove more efficacious.

With the progress of infectious disease, especially of a protracted form, there is a tendency to the development of an adynamic or typhoid state. This is to be met by careful selection of nourishment, which should be given in small quantities frequently; by alcoholic stimulants; the very free use of water, especially distilled water, which should be administered by every available avenue; by the most perfect rest and by the most carefully selected medicines, of which *arsenic*, *baptisia*, *rhus tox.*, *phosphoric acid*, and *hydrochloric acid* are the most important.

If drowsiness tends to pass into coma, it is a grave symptom, although its gravity can only be estimated by the associated symptoms. I once observed a case of typhoid fever in which the patient lay for sixteen days without recognizing friends, although the coexisting symptoms were not alarming. He was apparently soon relieved by *phosphoric acid*, which remedy and *lachesis* are the most important medicines for this semi-comatose state. For the graver cases *opium*, *hyoscyamus*, *hyoscine*, *stramonium* and *zinc* are more useful. Proper feeding should not be overlooked, nor the free use of alcoholics or other stimulants, such as coffee. In certain diseases, especially scarlatina, douches of cold water to the surface of the body, especially the head, neck and chest, sometimes afford the happiest result. All conditions of drowsiness, coma, and adynamic symptoms generally, call for an investigation of the secretory functions. The bladder should be daily inspected and the catheter used if necessary. Under these circumstances the bowels are apt to be forgotten. If the condition of the bowels permits, food-enemata may be administered three or four times daily. The existence of complicating conditions should be known at the earliest possible moment. This will be accomplished only as the result of frequent exhaustive examinations of the patient, interrogating every organ and securing proof of statements. Special attention must be paid to the lungs, heart, and kidneys. The frequency with which the lungs are attacked by pneumonic processes, by hypostatic congestion, œdema, or the bronchial tubes inflamed in the course of infective disease, is sufficient indication for daily investigation of these organs. The influence of position in the production of these processes is well known. The patient should therefore be frequently changed from the side to the back and from one side to the other. From time to time the patient should be encouraged to take a few deep inspirations, even if cough and accelerated breathing result, for in this manner the remote portions of the lungs are inflated and accumulations of mucus within the bronchial tubes dislodged. The influence of a feeble heart in the production of pulmonary complications must not be overlooked.

**FAILING HEART.** Death is very frequently the immediate result of a failing circulation. This fact has received so much attention within recent years that undue importance has been attached to this symptom, even the laity making frequent use of the much-abused term, "heart-failure." In order to a proper application of this term in febrile diseases the degree of failure in heart power must be out of proportion to the general failure, *i. e.*, it must be the most prominent clinical feature of the case. With failing heart there is usually a rapid pulse with increasing feebleness and often disturbance of rhythm. Physical examination of the heart will reveal gradual elimination of the muscular element of the first sound, with a resulting first sound approximating in character to the second sound. The importance of early detection of heart-failure



which is out of proportion to the general failure is therapeutically very important. But only the practitioner who habitually studies the state of the circulation in fevers can be trusted to give an intelligent opinion.

**THE SKIN.** In these days of frequent spongings with cold water proper cleansing of the skin with soap and water is neglected. If the patient lies much in one position the parts which bear most pressure are liable to inflame, ulcerate, or slough. This is favored by the feeble circulation. Frequent change of position, scrupulous cleanliness, and astringent washes may arrest the process. After the formation of an ulcer zinc ointment may be applied, making use of a preparation which will not melt at body heat, and over this a protective dressing held in position by adhesive strips. Such ulcers should be cleansed at least twice daily and bathed with an antiseptic solution. Various affections of the ear, eyes, bladder, etc., call for attention more frequently during the period of convalescence. For the care of these, the reader must consult special treatises.

**CONVALESCENT PERIOD.** The character of treatment accorded to a convalescent from infectious disease varies so much with the nature of the affection that only the most general instructions can be formulated in this place. The most frequent error committed at this time is in allowing patients to resume too rapidly their ordinary habits of diet, exercise, recumbency, etc. When one considers the degree of tissue degeneration taking place in the course of fever attacks, it is easy to understand that time is a most important element in restoration to a normal condition. Premature exertion leads to a retardation in recovery and sometimes to permanent crippling of an organ. Not only should mental and physical exertion be slowly resumed, but the digestive apparatus should not be too rapidly taxed. Solids should be slowly resumed and the influence of new articles carefully watched. The nutritive process is stimulated by means of gentle massage, cool sponging, and faradism. Change of air is frequently beneficial, and in general it is best to keep the patient in the open air as much as the weather will permit. The sequelæ of infectious fevers are numerous and should be carefully watched for and treated. The frequency of their occurrence is greatly diminished by good treatment of the primary affection.

## TYPHOID FEVER.

**Synonyms.**—Enteric fever; pythogenic fever (Murchison); typhus abdominalis (German); dothiéntérie (French).

**Definition.**—A specific continued fever, lasting usually from three to four weeks, pursuing a definite course, and characterized by a peculiar eruption, intestinal lesions, diarrhœa, and enlarged spleen.

**History.**—The recognition of typhoid fever as a separate morbid entity is an advance of the present century. That the disease existed and cases of it were described at more remote times, we have abundant evidence to prove. In John Hunter's museum are two specimens showing the characteristic intestinal lesions. Spigelius in the seventeenth century described cases of a continued fever with intestinal lesions. Typhoid fever is undoubtedly the same affection as was described by Gilchrist, of Dumfries, in 1734, under the name of slow nervous fever. That prominent clinicians even in those days were not inclined to class all continued fevers under the one head, is shown by the fact that Huxham, in 1738, took care to differentiate the "nervous fever" of Gilchrist from "putrid malignant fever" (typhus). At still more remote periods, when malarial fevers were wider spread and correspondingly more severe, typhoid fever was classed among the remittent fevers. Baglivi, in 1696, and Lancisi, in 1718, described cases of fever in Rome in which the intestines were ulcerated.

In 1813 began the movement which led to the separate recognition of the different continued fevers. In that year, Bretonneau, of Tours, described the disease under consideration, separating it from typhus and calling it "dothiéntérie." Pettit and Serres later described it as meso-enteric fever. The name typhoid fever was given the disease in 1829 by Louis in his great work on fevers. Gerhard and Pennock, of Philadelphia, writing in 1837, thoroughly established the identity of typhoid fever from typhus. The question was finally set at rest by Sir William Jenner, whose investigations, published in 1851, settled all doubts which had hitherto existed in England.

**Etiology.**—At this late day it seems unnecessary to present evidence to show that typhoid fever is a specific form of fever developed from the action of a specific poison upon a susceptible system, as the general consensus of opinion is now overwhelmingly in favor of the doctrine that typhoid fever is due to a specific germ; yet there are still a few prominent physicians who do not accept this view. Murchison, with his ability and the prestige gained by his great work on fevers,

occupying a prominent position as an opponent of the germ theory as related to the specific fevers, advocated under certain circumstances the spontaneous origin of typhoid fever. Apparent evidence in favor of such a doctrine may occasionally be found; as witness the occurrence of typhoid fever on an island in the Pacific Ocean where no case of that disease had existed for over a year; and the recent severe but limited epidemic at the Chester Military Academy, an explanation of which has never been given.

The professional position regarding the disposition of typhoid fever patients in hospitals is the strongest evidence of a general disbelief in the contagiousness of this fever, they being now freely admitted to hospitals, and unhesitatingly placed side by side with patients suffering from other diseases without fear of spreading the disorder. The possibility of a slight degree of contagion was entertained by so good an observer as Sir William Jenner, who stated that in the days when clinical thermometers were not made self-registering, some students and others who were frequently stooping over typhoid patients to read the registration of the fever, contracted the disease. A belief in a limited contagion is, we think, warranted by facts unnecessary to detail at this time. The origin of the poison of typhoid fever is in the human body; it being eliminated mainly through the stools. Possibly the poison is also eliminated by the breath, but to a very slight degree. It has also been claimed that it is given off by the emanations from the surface of the body, but much of the evidence tending to show the communicability of typhoid fever by the breath, perspiration, urine, etc., is open to criticism. After the expulsion of the poison from the body, a certain period of time elapses before the specific element possesses the power of communicating the disease to others. Nearly all observers agree that the poison of typhoid fever requires a residence for a period of time, as yet uncertain, in decomposing organic matter outside the body,\* before it is capable of communicating the disease to others. A soil saturated with human excrement is an important factor in the propagation and spread of the germs. Such a soil may be accidentally inoculated with the stools from a single case, and thus become a veritable culture-bed, with epidemic results. Aside from the influence of such a soil in causing a rapid development of the germs, a permanent culture-bed remains, frequently infecting water supplies, and becoming a more or less constant source of infection. In large towns and cities where the paving and drainage are poor, soil soakage is extreme, and permanent sources of infection are developed. Privies, by poisoning the water supplies through the medium of the earth's strata, are a prolific source of infec-

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\* Cayley thought that stools contained in the pans in the closets of Middlesex Hospital communicated the disease to other patients within twenty-four hours.

tion. In some cases there has been discovered direct communication between the privies and the water supply. The recent Plymouth (Pa.) epidemic was one of the most remarkable instances of the infection of water supply by typhoid stools. In this town of 8,000 inhabitants, there occurred 1,500 cases of typhoid fever, the epidemic being limited to a section obtaining its water supply from a contaminated source. Investigation revealed that one typhoid fever patient whose disease was contracted in Philadelphia, in a house where there had been numerous other cases within a comparatively short period, was responsible for the outbreak. Coming to Plymouth, he was taken ill, and his stools were emptied on the ground near the banks of the stream from which the water supply was obtained. When the spring thaw came, the excreta were washed into the stream, thus contaminating the reservoir, and the epidemic followed.

Next to water, milk is the most prominent carrier of contagion. Pure milk, *per se*, does not seem to be capable of conveying the disease. It is a generally accepted view that this article of diet is only dangerous when mixed with infected water, accidentally present from failure to cleanse cans which have been washed in it, or added for purposes of adulteration. When the germs do contaminate milk, they are especially dangerous because they multiply very rapidly in that fluid.

Of the various solid foods, meat is the most likely medium of transmission of the poison. It is probable, however, that the meat must be in a state of partial decomposition, furnishing thus the organic material under conditions favorable for the development of the typhoid germ. One of the most interesting illustrations of this latter method of dissemination of the disease, was the recent epidemic at Klotten, near Zurich, 668 persons developing the disease after partaking of infected meat.

Atmospheric spread of typhoid fever is limited. Many do not believe in the aerial spread of the poison at all. There can be no doubt, however, that such a transmission of the disease occasionally occurs. Especially is this liable to occur in hot, foul, illy-ventilated apartments. In such cases the bedclothing is infrequently changed, carpets are foul and probably contaminated by improper handling of excreta. The possibility of contagion by the breath was shown conclusively by Sicard, who caused typhoid fever patients to breathe several times through curved tubes into water that had been boiled and sterilized. He was thus enabled to demonstrate the existence of typhoid bacilli in the expired air. Osler, however, is very strong in the expression of his opinion that in cases arising by contagion the poison is not given off by the breath but by the fæces. In dry, dusty seasons, it is believed that the poison may be carried about in the dust by winds. A free supply of oxygen renders the germ inoperative, as is shown by the innocuousness of properly conducted sewage farms.



Age is a powerful predisposing factor in the etiology of typhoid fever. It is uncommon in advanced age, and at one time was believed to be very exceptional in childhood and infancy. But recent observations indicate that typhoid fever is more frequent in young children and infants than has been hitherto supposed. "Gastric fever," "infantile remittent fever," etc., are terms which we are warranted in frequently interpreting as typhoid fever. We may state in a general way that the frequency of the disease in relation to age seems to increase with each year up to adult life. The average age in two hundred cases treated by the author in private practice was twenty-one years and eight months. After the age of thirty years, there is a rapid falling off in the relative number of cases. Histological investigations by the writer into the structure of the mucous membrane of the bowel and of the involved glandular structures at different ages, promise results throwing some light upon the frequency of the disease in early life. Pepper suggests that the failure of those of advanced years and middle life to contract the disease may find an explanation in an acquired immunity from constant exposure to the poison, or that the majority of those possessing personal susceptibility acquire the disease in early life.

Individual susceptibility is undoubtedly a very important factor in the causation of this fever. So widespread is the poison that there can be but little doubt that very few persons can avoid taking it into their systems at some time in their career; yet comparatively few yield to its baneful effects. Young men and women who are newcomers in a community in which typhoid fever prevails, seem especially liable to contract it. Such immunity is lost, however, if the subject removes to other places. The possession of a healthy gastro-intestinal tract is undoubtedly a very important factor in resisting the entrance of the poison.

The sexes seem to be about equally affected. Hospital statistics, however, show the contrary, that males are more frequently affected. This discrepancy is explained by the greater frequency with which the male sex applies for hospital care.

SEASON AND CLIMATE. Typhoid fever is universal in its distribution, although it is especially frequent in the temperate zones. Still, it is not uncommon in the tropics. Cases have likewise been observed in countries as far north as Iceland, Norway, Sweden and Finland.

The name, "autumnal fever," so frequently used, indicates the greater prevalence of the disease during the fall or autumn months. The frequency by months, however, is affected by the zone in which the disease occurs. The further south we go, within moderate limits, the earlier in the year is the highest number of cases reached. In the south temperate zone the disease develops quite early, attaining its maximum number of cases in June or July; while further north, September or October gives the greatest number of cases. It is especially liable to

prevail in the fall months, when a hot, dry summer has preceded. The explanation offered for this observation is that under such circumstances the water supply is low, and any poison contained therein is in greater concentration. The general standard of public health is more likely to be poor at such times.

In Philadelphia, as in other large cities, the disease has gradually assumed an endemic character, and we now have typhoid patients on our visiting lists during every month of the year. This is less noticeable in cities further north.

We have abundant evidence at our disposal to show the prevalence of typhoid fever in certain families, and the immunity of others. Parents who have suffered from the disease may fear its ultimate appearance among their children.

REPETITION. Typhoid fever protects against itself less frequently than is the case with most of the acute infectious diseases. Many persons within the acquaintance of most of us have had the disease more than once; and a small percentage of persons have had it repeatedly.

Certain diseases, such as phthisis, and certain conditions, such as pregnancy, are, in a measure, protective.

ATMOSPHERIC CONDITIONS. Statistics show the greater prevalence of typhoid fever after hot, dry periods. In the north temperate zone a hot, dry summer is followed by an increase of the disease towards the fall. Murchison considered a warm, moist atmosphere, with a slight rainfall, as most favorable to its spread.

Typhoid fever does not respect persons or localities, but attacks or invades all; rich or poor, feeble or robust, city or hamlet; no one is exempt. It is the most universally present and constant of any of the group of infectious fevers.

**Pathology and Morbid Anatomy.**—**ESSENTIAL CAUSE OF TYPHOID FEVER.** The predominating views respecting the causes of the group of acute infectious fevers have led to painstaking care upon the part of bacteriological investigators to discover the micro-organism supposed to possess a causative relation to typhoid fever (Koch, Eberth, Friedländer, Gaffky, Fränkel, Simmonds and W. Meyer). As the result, a bacillus has been discovered, which exists in the body only during the course of this disease. The breadth of this bacillus is about one-third of its length; compared with a red blood corpuscle, its length is about one-third of the diameter of the latter. Its extremities are rounded; certain appearances of these bacilli, in the centre, have led to a difference of opinion as to whether they contain spores or vacuoles. In rapidly growing cultures many of the germs adhere at their ends, forming thus threads of bacilli. Cultivations can be readily carried forward upon gelatine beds at temperatures of 65° F. and upwards. The colonies appear as whitish or bluish-white, irregularly contoured islands. The

cultivating medium remains solid. It is interesting to watch its growth upon a boiled potato base, which is that of an invisible cuticle, becoming visible, however, if the potato be made alkaline in reaction. When examined in fluids the bacilli manifest considerable activity. The intestinal canal contains the largest number of this organism. Early in the disease they are found to have penetrated the lymphatic system of the abdomen, being found even in the mesenteric nodes and the spleen. The blood, liver, kidneys, lungs, and, in fact, most tissues, have been found to contain them.

Koch's recent statement made before the International Medical Congress, Berlin, 1890, is important in this connection. He said that a very characteristic example of the difficulty with which the determination of a bacterial species is surrounded is exhibited by the bacillus of typhoid fever. If this organism be found in the glands of the mesentery, in the spleen, or in the liver of a typhoid patient, there can be no question but what we are dealing with the real typhoid bacilli, since no other bacteria have hitherto been found in these regions which could be confounded with them. But when we are dealing with the determination of typhoid bacilli in the contents of the intestine, in soil, in water, or in dust, the circumstances are very different. In such places there are found numerous bacilli similar to those of typhoid, which only a very skilful bacteriologist, and even such an one not with absolute certainty, can distinguish from the typhoid bacillus, as unmistakable and constant characteristics of the latter are, as yet, wanting.

There can be little doubt that the immediate cause of pathological change and the resulting symptoms is a ptomaine, the product of developmental changes taking place in the bacillus. Inoculation experiments upon animals have been highly corroborative, the artificial disease resembling the natural one as nearly as might be expected. The frequency with which this germ is found in drinking water renders it practically important to know that freezing does not destroy it. Prudden found them still capable of activity in ice. The boiling temperature is, however, thoroughly efficient for their destruction. They may be destroyed in bouillon cultures by carbolic acid solution, 1 : 200, or by corrosive sublimate, 1 : 2,500. The life-history and pathogenic action of the bacillus of typhoid fever is not yet well known in all its details; but enough is certain to make us quite confident of its relation to the disease, *i. e.*, as its active specific cause. For its discovery the staining method of Ziel\* is as good as any. Thus far efforts to cultivate it from the fresh intestinal discharges have been rather unsatis-

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\* Filtered saturated solution of carbolic acid, 90 parts; saturated alcoholic solution of fuchsine, 10 parts. Stain for one-half hour; remove excess of coloring matter with alcohol; clear with oil of cedar and mount in Canada balsam.

factory. Investigations to determine the life-habits of this germ, without, as well as within the body, and to make clear the avenues of entrance, are of the greatest importance to preventive medicine.

The tissue changes occurring in typhoid fever will be considered under the following heads: (1) Those peculiar to or characteristic of typhoid fever; and (2) Those common to acute infectious fevers generally. The specific lesion consists of inflammatory changes occurring in Peyer's patches\* and solitary glands of the intestines, most marked in the lower portion of the ileum, due to the predominance of gland structure at this point; although typhoid ulcers are common in the cæcum, and are occasionally discovered beyond and behind this point. A very convenient method of the study of these changes relates to their progress during each week of the disease.

*First week.* During this period there is observed swelling with, perhaps, commencing ulceration.

*Second week.* The ulceration continues, and before the end of the week a certain amount of sloughing takes place, and is manifested in a number of the inflamed centres.

*Third week.* The sloughing progresses, leading sometimes to coalescence of adjacent lesions, or perforation, or both.

During the *fourth week* ulceration and sloughing may be still extending in some of the inflamed glands. Many of them, however, manifest the healing process, during which the swelling diminishes and the ulcers clear and present granulating surfaces. The further time required for the healing of the ulcers depends upon many circumstances, such as the observance, or not, of a proper diet, the amount of exercise taken, and the tissue activity of the patient; cases apparently identical, as far as we are able to judge in the present state of our knowledge, leaving the subjects in widely differing degrees of nutritive activity. It must not be forgotten that the pathological changes do not always progress in parallel lines, even in neighboring lesions, for the reason that the inflammatory focus is so much the more active in some; and, again, the changes begin at a much later date in certain of the solitary follicles than in others. One finds, therefore, as neighbors, a sloughing gland and one but slightly ulcerated, and still another merely swollen. The base of the ulcer, or separating slough, may consist of any of the coats of the intestine. The larger diseased patches are oblong, and, unlike tubercular ulcers, develop parallel with the length of the intestine. Occasionally the inflammatory changes extend to the adjacent tissues, and very extensive ulceration results, even so great as to surround the

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\* This has been denied, the denial being based on the observation of certain few cases presenting during life all the phenomena of typhoid fever and in whom autopsy failed to show the intestinal alterations. Other pathological changes, as enlarged spleen, and the presence of the typhoid bacilli, were present.



intestinal tube. Such widespreading ulcerations are seldom found excepting in the neighborhood of the ileo-cæcal valve. The smaller patches and solitary follicles develop ulcers with a circular outline. All swellings are soft, and of a yellowish-gray color, unless stained by bile, when a brownish tint is imparted to them. Patches intensely inflamed are firmer. Cicatrized ulcers may show a grayish, "slaty" discoloration for years. Exceptionally the lesions may be observed characteristically developed in the large intestines, as in three cases recorded by Laveran.

Varying degrees of intensity of catarrhal inflammation of the intestines attend all cases of typhoid fever and are responsible for many of the symptoms. If well marked, the entire mucous surface is puffy and dark red.

**LYMPH NODES.** The mesenteric nodes manifest alteration very soon after the primary intestinal inflammation is established, the degree of change possessing a close relationship to the intensity of the latter. Foci of suppuration are common, undergoing absorption or encapsulation in cases of recovery. Rarely the inflamed node excites local or general peritonitis.

**THE SPLEEN.** All well-developed cases of typhoid fever are attended by marked splenic enlargement. A skilful physical examiner can often detect it by the end of the first week of the disease. The swelling begins early and continuously increases for fifteen or twenty days. It then remains stationary for two or three days longer, when it begins to decline. The organ is dark in color and firm in its texture until the maximum size is reached, when it softens. Hyperæmia and increase of the normal elements with dark brownish pigmentation are the causes of the enlargement. Infarctions and hæmorrhage into the peritoneal cavity from rupture are occasional.

The **LIVER** is often found softened, and occasionally the hepatic elements are in such an advanced stage of granular and fatty degeneration that their outlines are lost.

The **KIDNEYS** are often the seat of parenchymatous inflammation, sometimes of infarctions.

**VASCULAR SYSTEM.** Degeneration of the muscular fibres of the heart is quite constant. Generally the heart-muscle is soft and flabby, tending in color to gray or brown. The microscope exhibits infiltration of the muscular elements with minute granules or brown pigment, or both. Hyaline degeneration is less frequent, the tissue then being firmer, brittle, and glistening upon its cut surface. Thrombi and vegetations are occasionally found within. Their detachment sometimes leads to embolism and its consequences.

**ARTHRITIS** may be developed, especially during the early period of convalescence. Late in the course of the fever thrombosis of the veins sometimes occurs, the femoral vein suffering oftenest.

**RESPIRATORY TRACT.** The larynx is frequently catarrhal. Superficial or deep ulcerations may ensue. Catarrhal inflammation of the larger bronchi is almost constant; capillary bronchitis is rare. Bronchopneumonia is quite frequent and presents the usual varieties. Hypostatic congestion is quite constant and is sometimes associated with hepatisation. Typical lobar pneumonia is rare.

**OTHER GLANDULAR STRUCTURES.** The parotid gland is rarely the seat of an infective inflammation tending to suppuration. Orchitis and ovaritis are very unusual indeed. The pancreas has been found swollen, red, and, later in the disease, it may become firmer and of a grayish or yellowish hue.

The general muscular system is often the seat of hyaline degeneration. The tissues of the skin, mucous membrane, and the connective tissues generally are all subject to the varieties of inflammation and their consequences due to the altered blood infection, pressure, position, etc.

**NERVOUS SYSTEM.** Meningitis, thrombosis, embolism, arthritis, and neuritis are all rare. General anæmia and perverted nutrition of nervous tissue are, of course, constant features of the disease, and are responsible for many of its classical symptoms.

**Clinical Course.**—The duration of the stage of incubation in typhoid fever is at present undecided. It is a general belief, however, that it lasts about two weeks. Cases have been observed in which it lasted a much shorter time. Instances in which the incubation has been short are apparently those in which the poison has been introduced into the body in a rather concentrated state. Delay until four weeks after acknowledged infection has also been reported. This latter fact must not lead to the positive statement that these four weeks represent the entire incubation period, for while the typhoid poison may have been introduced into the alimentary canal at the time believed, it may not have entered the system to begin its toxic work until much later.

An attack of typhoid fever is usually preceded by a well-marked prodromal period, which represents the interval during which the specific causative element is attacking the organism. Its duration ranges from two or three to twelve or fourteen days. Headache, malaise, anorexia, possibly constipation, and near the close of the period, slight chilly sensations, are the symptoms most frequently complained of. Although these constitute the main symptoms of the prodromic stage, there are others, more characteristic though less frequent in their occurrence—partaking largely of a neurotic nature. They are mental confusion, insomnia, general prostration, and dulness of hearing. There may also appear epistaxis and slight bronchial cough. Pepper states that he has many times been led to anticipate typhoid fever by the unusual dulness of hearing and occipital headache coming on after a few days of general

malaise. This prodromic period has been so much dwelt upon by medical authors that it has come to be considered an essential feature of an attack of typhoid fever. On account of this general belief I desire to lay especial stress upon the statement about to be made that many cases begin abruptly, the first symptoms that appear being such as belong to the stage of invasion. This sudden advent of the disease is far more apt to occur in children than in adults.

In the clinical study of typhoid fever one is very apt to place a value on certain symptoms according to the period of the disease in which they occur. Care is therefore observed to chronicle the number of days the fever has already lasted. This is not always an easy matter by any means, for it is very rare indeed that the patient comes under the physician's care at once on the incidence of the disease; and very often the early symptoms are of such an indefinite character, that unless the thermometer is used in all cases as a routine measure, the nature of the trouble is very apt to be overlooked. It is therefore a very difficult matter to say exactly when the disease began.

**FIRST WEEK.** The stage of invasion, including the first week of the disease, begins with elevation of temperature, and increase of many of the symptoms complained of during the prodromic period, to which are added soreness of the muscles of the body, backache, epistaxis, and a variety of symptoms indicative of disorders of the gastro-intestinal tract, such as furred tongue with red edges, thirst, nausea, sometimes vomiting, and toward the close of the week, more or less tenderness and puffiness of the abdomen. The tenderness is more marked on the right side of the abdomen, and pressure in this region will often develop gurgling. During the first week, the temperature rises steadily, each successive morning and evening temperature being higher than those of the preceding day. The fever generally reaches 102.5° F. to 104° F. by the fourth or fifth day, and by the seventh or eighth day, the maximum degree of fever is likely to have been attained. The pulse is increased in frequency, but generally not in proportion to the extent of the elevation in temperature. It is full, of low tension, and generally dicrotic. This last characteristic is regarded by many as significant of the disease. The bowels are usually constipated, but diarrhoea may supervene at any time, few cases passing the eighth day without this symptom. The stools are loose, gruel-like in consistency, often frothy, and of a yellowish or greenish color.

**SECOND WEEK.** During the second week the temperature chart will exhibit comparatively slight fluctuations, the morning remissions amounting to from one to two degrees Fahrenheit. The frequency of the pulse increases, and it loses its dicrotism. With the full development of the disease, which occurs during the second week, the patient's appearance becomes quite characteristic. The countenance loses its usual brightness,



the eyes are suffused, the cheeks show a red flush, and the lips are dry. At this time a careful inspection of the abdomen will usually result in the discovery of the specific eruption of typhoid. This eruption is usually discoverable as early as the seventh day of the disease; and it may be delayed until the fifteenth day. In a very small percentage of cases it may be altogether absent. Enlargement of the spleen may exceptionally be detected as early as the fifth day, but is clearly distinguishable in the majority of cases by the middle of the second week. The diarrhœa which usually ushers in the second week of the fever, continues throughout the disease, the stools varying in frequency and character. Distension of the abdomen in slight degree is quite constant. Aggravated tympanites is rare. The urine is scanty, abnormally high-colored, and sometimes contains small quantities of albumin. In the majority of cases a mild form of delirium supervenes during the second week. This delirium may assume a violent character. After this symptom once comes on it seldom disappears until the termination of the illness.

**THIRD WEEK.** During the third week, if the tendency of the disease is favorable, the daily remissions of temperature become more marked, and the maximum fever of the day a little lower. Marked symptoms of convalescence are postponed until the fourth week. The rapidity of the pulse, however, does not at once diminish with the fever. The heart's impulse is feeble. The typhoid state is now fully developed. The patient's general condition, as to weakness, etc., has increased over that of the second week. In aggravated cases the prostration may be great, emaciation extreme, the tongue dry, brown and cracked, it and the teeth being covered with sordes, the heart exceedingly feeble, the stools frequent and often involuntary, the tympanites extreme, and hæmorrhages from the bowels repeated. The delirium may become more violent in character, passing into coma and associated with jerking of tendons and carphologia or some pulmonary complication. Any of the latter symptoms may unfortunately appear in the second week, if the case be a severe one. At this time the lowered vitality favors the development of bedsores.

**FOURTH WEEK.** In favorable cases the temperature is apt to register at the normal point in the morning. Afternoon or evening fever is often apparent for several days, and it is not uncommon for a subnormal morning temperature to be present for some days. Along with the remission in the fever there is a general subsidence of all symptoms.

**Analysis of the Symptoms.**—The FEBRILE SYMPTOMS of typhoid fever are particularly important. Observations of the temperature course in this disorder are absolutely necessary; we cannot make a correct estimate of individual cases without regularly repeated thermometric observations. The frequency with which such observations should be made will depend very much upon the character of the case. There



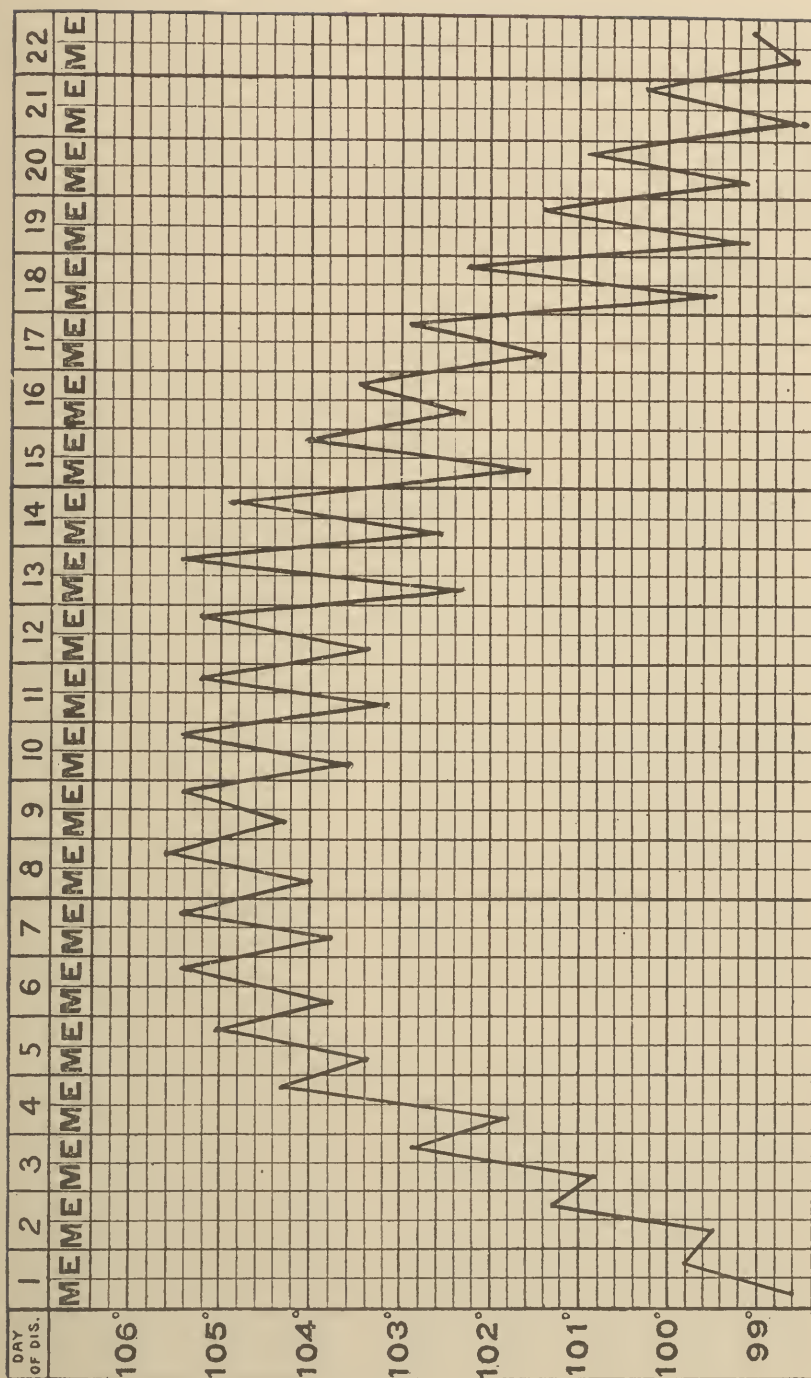


FIG. 1.—THE GENERALLY ACCEPTED TEMPERATURE CURVE OF TYPHOID FEVER, WITH MORNING REMISSIONS AND EVENING EXACERBATIONS, THE FEVER RISING AND DECLINING BY STEPS. (AFTER WUNDERLICH.)

should be at least two observations daily in all cases, however light, and these should be made preferably in the morning and evening. Periods for the more frequent taking of the temperature will suggest themselves to the practitioner, according to the severity of the illness. The temperature should be taken by preference in the rectum, surface observations not being always reliable. When alarming symptoms are present, the temperature must be taken and recorded frequently, even if the ordinary evidences of high fever are not apparent. In other words, do not trust appearances; trust only the thermometer. The temperature varies considerably during the course of the disease. The opportunity for making temperature records during the onset of the trouble rarely presents itself, for patients, during the initial period, are seldom under the care of the physician, or if so, the serious nature of the ailment is not sufficiently clear to suggest to the medical attendant the necessity for thermometric observations. This initial period, as already stated, embraces some three or four days, during which the temperature frequently rises in the manner originally described by Wunderlich, and copied extensively by medical authors. That is to say the temperature rises by steps, the respective morning and evening temperatures of successive days being higher than those of the preceding; each morning temperature, however, being lower than that of the preceding evening. On account of the widespread belief that the temperature curve, as described by Wunderlich, is present in most cases of typhoid fever, it is important to note the frequent exceptions observed, especially by American physicians. According to the observations of the author, the typical rise of Wunderlich is the exception rather than the rule. Personally I have noted in quite a percentage of cases a very rapid rise of temperature at the onset, preceded or not by a day or two of slight febrile action, such rapid rise not being attributable to any discoverable complications. By the close of the second week the temperature will usually have reached its maximum, the morning remissions and evening exacerbations presenting very slight variations. During the third week the morning remissions are more marked, the maximum evening temperature of the second week, however, being continued through the third week. In the latter part of the third week the morning remissions become more pronounced daily. The feature of the temperature of the fourth week is intermittency. The evening rise shows daily reduction until the normal is reached. At this time a subnormal temperature is common, sometimes continuing for one or more weeks. Recurring elevations of temperature during the fourth or later weeks are the result of complicating conditions, and are no part of the essential fever, excepting it be a relapse. A rapid rise of temperature during any period of typhoid fever, excepting, perhaps, in young, excitable subjects during the onset of the disease, indicates a serious condition. A rapid fall rarely occurs before the latter part of the second

week, and if it does occur, it is suggestive of intestinal hæmorrhage, which may be concealed or apparent in the stools; of perforation, or it occasionally attends rapidly occurring heart-failure. A gradually increasing morning remission indicates a favorable course. On the contrary, a very rapid increase in the morning remissions may be followed by subsequent elevations. The temperature is usually low during the earlier days of the first week, seldom going beyond  $100^{\circ}$  F. to  $102^{\circ}$  F.; during the latter part of the first week its range is from  $102.5^{\circ}$  to  $104^{\circ}$  F. Occasionally, even as early as the third day, the temperature may mount to  $105^{\circ}$  F. or higher. Such cases frequently lead to erroneous diagnoses. A very high early temperature is not necessarily followed by a correspondingly high temperature later, for such a condition may occur in cases of ordinary severity, or again, it may mark the beginning of a very severe attack. Neurotic individuals are especially liable to hyperpyrexia, the excessive fever being maintained for days without any other evidence of any complication whatever. Under such circumstances the fever possesses no special significance. Per contra, we meet with phlegmatic indi-

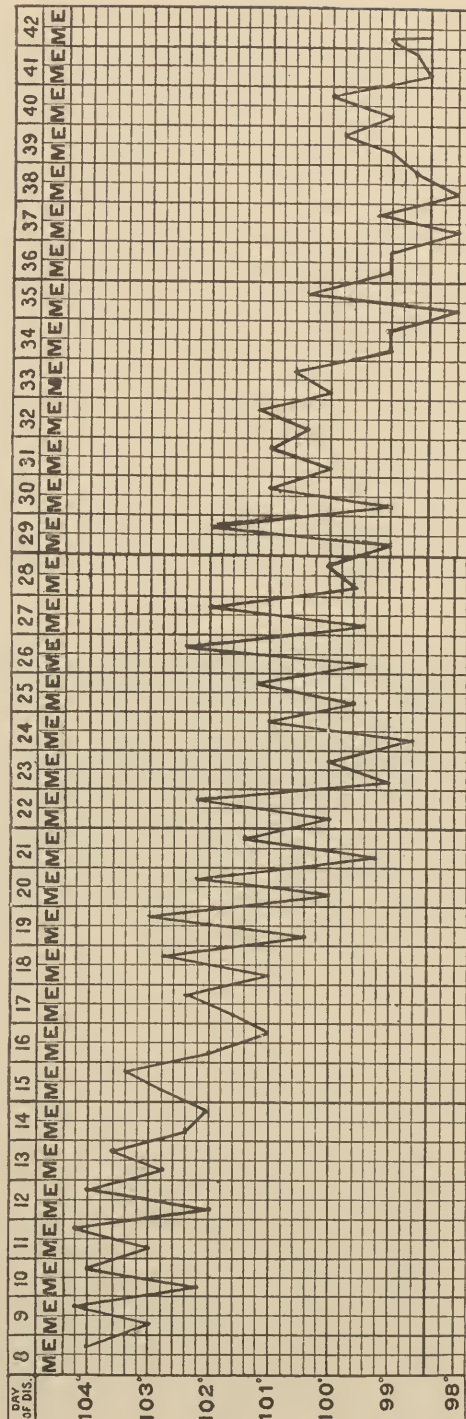


FIG. 2.—TEMPERATURE CHART OF A PROLONGED CASE OF TYPHOID FEVER, IN THE HAHNEMANN HOSPITAL.



viduals in whom, notwithstanding the many serious conditions present, the elevation in temperature is at the most but moderate. The temperature range at the close of the first week is a pretty fair guide to the probable elevation, which will be observable during the second. The second week is the week of full development of the disease, and by the latter part of this period, at least, the maximum temperature is reached. In active cases we seldom have a morning temperature of less than  $103^{\circ}$  F., or an evening temperature below  $104^{\circ}$  or  $104.5^{\circ}$  F. The height to which the fever attains during the second week is usually retained during the third. It sometimes happens during the latter period that the temperature rises suddenly to a great height. Still, in typhoid fever it does not attain the point of  $105^{\circ}$  F. or over with anything like the frequency it does in typhus, scarlatina and other acute infectious fevers. It must, however, be regarded as a serious symptom, be the attending circumstances what they may. The gravity of the high temperature is much lessened if it be interrupted by remissions; the longer and the greater the remissions, the more favorable being the outlook. During the fourth week the temperature falls by lysis, unless kept up by complicating conditions.

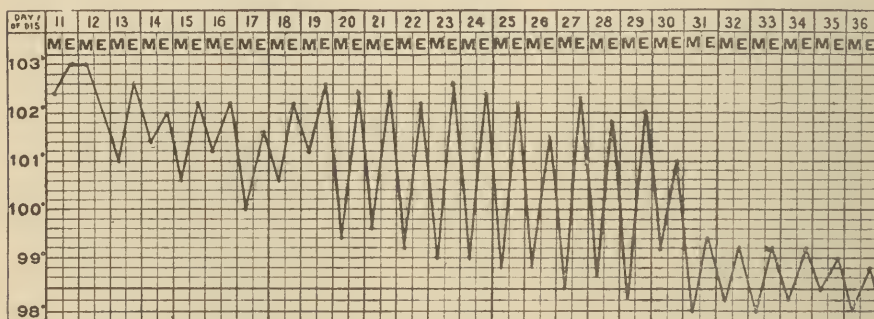


FIG. 3.—TEMPERATURE CHART OF A CASE OF TYPHOID FEVER, TERMINATING AS AN INTERMITTENT FEVER.

While the above remarks comprise a description of the temperature of typhoid fever, as observed in nearly all cases, there are some most remarkable exceptions which must be borne in mind. The knowledge of their possible existence is sufficient to guard against error. The inverted type of fever is sometimes met with. In this the exacerbations of fever occur in the morning, and the remissions in the evening. These phenomena are observed more particularly in children. Sometimes the temperature drops very suddenly to the normal. This condition is one of great concern, for it may mark the beginning of an attack of pneumonia or the occurrence of intestinal perforation or hæmorrhage. Very anomalous cases, characterized by entire absence of fever, have been reported from time to time.



In some cases the temperature curve of a case of typhoid fever assumes the characteristics of an intermittent fever toward the close. This condition is held by many physicians to indicate a malarial complication. The only proof of the malarial nature of this feature is obtainable from a microscopic examination of the blood. If the organisms of Laveran are found, then the existence of malarial poisoning is unquestionable, but not until then.

**CIRCULATORY SYMPTOMS.** A somewhat definite parallelism exists between the height of the temperature and the rapidity of the pulse; still many cases will be found in which the pulse is but little accelerated, although the temperature is high. With the early rise of fever, there is an increase in the frequency of the pulse, the latter reaching 100 or 110 to the minute by the close of the first week. During the second week the pulse creeps up a little, manifesting only a slight increase over the first week. In the third week, the continued fever, increasing debility and cardiac degeneration, cause considerable rise in the pulse-rate, the latter often reaching 130 to 160, thus losing the parallelism that had hitherto existed between it and the temperature. Such a great frequency of the pulse must be regarded as of very serious omen; some few cases, however, recover. While the high pulse-rate of the third week is usually attended by high temperature, there are notable exceptions, due to the cardiac degeneration and general exhaustion to which reference has already been made. Irregularity of the pulse is not unusual after the middle of the second week. If well marked, this symptom becomes one of great gravity. Diastolic murmurs occur with such frequency that many have considered this peculiarity of diagnostic value. Many of the ominous symptoms of typhoid fever are the direct result of cardiac failure and its consequences, as cardiac thrombi and emboli in the important organs, pulmonary stasis, and pulmonary œdema. The weak heart leads not only to pulmonary emboli, but also unquestionably to the so-called hypostatic congestion. I have never seen a case of hypostatic congestion in the second week of the fever without preceding weak heart. Thrombosis, especially of the vessels of the lower extremities, occurs sometimes. A single limb is usually attacked, and the obstruction is attended by œdema. Dropsical swellings of the lower extremities are not infrequent at the close of the disease, due partly to weak heart and partly to the altered blood. From the very onset of the disease, careful observations should be made of the pulse, and also of the heart itself, as to their force, frequency and rhythm, etc. Failure of the heart is sometimes an early condition, and is not infrequently the result not alone of existing typhoid fever but of previously existing cardiac degeneration. The cardiac weakness is shown by decrease in intensity of the first sound of the heart. The hand and the ear should both be used in noting the condition of the organ. It is not wise to trust alone to the condition of the radial pulse.

Observation of the pulse continues interesting during convalescence. With increasing strength the pulse diminishes in frequency, and this may go on to such an extent as to become abnormally slow, but is no cause for alarm. In other cases the pulse remains exceedingly irritable for weeks and months, sometimes the slightest exertion causing it to become very rapid. This symptom calls for increased watchfulness over the patient, for it is often the only evidence of a cardiac degeneration.

**NERVOUS SYMPTOMS.** The nervous phenomena of typhoid fever occupy so prominent a place in the symptomatology of the disease that they led to the introduction into medical nomenclature of one of the fever's synonyms, "nervous fever." Headache is the earliest of the nervous symptoms to appear, it often ushering in the disease. Cephalalgia is present in nearly all cases, and its severity is by no means parallel with the severity of the attack, for it is sometimes very aggravated in mild cases. The pain is usually dull and heavy in character, and involves especially the forehead and temples, and occasionally the occipital region. Sometimes it assumes a neuralgic character, thus occasioning intolerable suffering. In one case which I saw recently, the pain was so agonizing as to suggest to the attending physician a diagnosis of meningitis. The pain increases in severity with the development of the disease, giving way, apparently, before the cerebral depression that gradually takes place. There is sensitiveness of the special senses, the intensity of the hyperæsthesia bearing a close relation to the severity of the headache. The patient also complains of pain in the back, pain in the limbs, and restlessness. Mental symptoms are present at some time in the majority of cases of typhoid fever. Still some remarkable exceptions to this rule are observed. Thus in two cases, sisters ill at the same time, the attacks were chiefly remarkable for the freedom from nervous symptoms of all kinds, notwithstanding the high temperature, which in one of them reached the unusual point of 106° F. Both patients made most excellent recoveries.

Somnolence more or less marked is present in nearly all cases at some period during the progress of the disorder, and usually develops early in severe cases. Delirium, which is always worse at night, is found in about two-thirds of the patients attacked by typhoid fever, and seldom appears before the latter part of the first week. At first there may be noted only a slight incoherency of speech after the patient arouses from sleep; he fails to answer questions correctly. This incoherence gradually grows worse and maniacal symptoms develop; the patient may try to leave the bed, endeavor to commit violence or jump out of the window. This tendency to get out of bed and wander about is by no means an unimportant matter for the physician or nurse, but is one which calls for the greatest watchfulness. It matters not whether

the delirium of the patient has hitherto been of the mildest and most harmless character, he should never be left alone, especially at night, lest it take a serious turn. He sometimes displays a recklessness that may result in serious injury, being dominated by some inexplicable impulse, probably fear. Later the low muttering delirium appears. The mental wanderings are generally connected with the daily occupations of the patient. It sometimes happens that the delirium develops with great rapidity very early in the case, indeed before there is the slightest suspicion of the existence of any physical ailment. These cases nearly always lead to diagnostic error, and often to the consignment of the patient to an asylum as one suffering with acute mania. After the delirium is once established it seldom disappears until the close of the disease. Automatic motions, such as picking at the bed-clothes, and grasping at imaginary articles in the air, with delirium, in many cases subsultus tendinum, are also frequent accompaniments.

Tremor is a common symptom of aggravated cases, and it is considered by some authors to be an indication of the extent of the intestinal ulceration. It is sometimes associated with tonic spasm of the limbs. Tremor is more apt to be present in those who have used alcoholic liquors, and in the young, and manifests itself especially in the hands, the tongue and the muscles of the face. Occasionally the entire surface of the body of a person suffering from typhoid fever is the seat of extreme hyperæsthesia, the slightest touch being painful. This condition is found oftenest in women of a hysterical temperament; if limited in extent, the lower half of the body is more apt to be affected. Cases have been reported in which the hyperæsthesia was sharply localized in the abdominal region, and when this is the case considerable care is required lest a diagnosis of peritonitis be made. The special senses show more or less disturbance in nearly all cases.

Interesting cases of *paralysis* are of occasional occurrence after the fever has run its course. They may present almost any distribution, and may be dependent upon disease of any portion of the cerebro-spinal nervous system. From brain disease they are dependent upon embolism, thrombosis, or extension of inflammation from the temporal bone; from spinal disease, upon poliomyelitis anterior or diffuse transverse myelitis; from the peripheral nerves, upon neuritis. Hemiplegia is observed most frequently during the third or fourth week. Most cases have terminated in complete recovery. It finds its origin apparently in a non-valvular lesion of the heart, leading to thrombi in the left auricle, whence emboli are detached.

Insanity has been observed as a sequel. Its development seems to be determined by an hereditarily unstable nervous organization, anæmia, the febrile toxæmia producing tissue changes, and the influence of the typhoid bacilli themselves.



*Meningitis* is rarely observed as the direct result of the typhoid poison, but usually occurs by extension of inflammation from neighboring structures.

**SIGHT.** There is sensitiveness to light, occurring more especially in connection with the accompanying headache, particularly if the latter be severe. The expression of the eyes is usually dull, but in some instances, particularly when marked delirium is present, the visual organs may present an unnatural brightness. Conjunctival injection may occur, but is rare. Some patients complain of obscuration of sight, not due, however, to a local eye complication. The pupils are generally dilated, but may be contracted, if violent head pains or stupor are present. Exceptionally, the pupils may display inequality.

**HEARING.** Deafness, even of high degree, is common. It often constitutes a prodromal symptom. It may later manifest itself during the fever, gradually increase, and if not due to organic causes, disappear during convalescence. It should never be forgotten that a certain proportion of these cases of deafness are due to otitis media, which is usually, however, unilateral. The majority of the cases of deafness in typhoid fever are due either to catarrh of the Eustachian tubes or the mental dulness of this disease. Ringing and buzzing sounds in the ears are commonly complained of. Large doses of quinine given by some practitioners occasionally induce deafness.

**TASTE.** Perverted taste is almost constant. With the full development of the disease, the altered state of the tongue and mucous membranes of the mouth accounts generally for this condition. Sometimes however, this symptom exists in the early part of the disease, with the tongue clean and moist.

**MUSCULAR SYSTEM.** Muscular prostration exists in all cases, and is especially severe in those of pronounced type. It is generally present from the first, growing worse with the progress of the fever; but in some cases it is noticeable early, before the disease has sufficiently advanced to produce profound tissue alterations, and in such, the prostration must be regarded as nervous rather than muscular. Late in the second week, and especially during the third, the muscular paresis is such as to almost warrant the use of the term paralysis, as is shown by involuntary stools and urine, and the inability to protrude the tongue. The same condition is often present in the intestines, as is indicated by the noisy rolling of fluids through the weakened channels. The patient lies upon his back, helpless, nearly motionless, and gravitates to the foot of the bed. Retention of urine may take place from paralysis of the cystic sphincter. Muscular twitchings and fibrillations are very common phenomena.

**THE LUNGS.** The rate of respiration varies with the pulse and temperature. Unusual rapidity of respiration is noted in consequence of tympanitic distension of the abdomen and from purely nervous influ-



ences. In some cases it is a manifestation of cardiac dyspnœa, resulting from degeneration of the heart muscle. Nearly all the inflammatory lesions affecting the pulmonary parenchyma and the bronchial tubes are liable to be found in typhoid fever at times. Bronchitis in some degree is constant, so constant indeed, that Stokes proposed that typhoid fever be called "bronchial typhus." I have very frequently found lobular lung solidifications, *i. e.*, areas of catarrhal pneumonia, oftener than is generally admitted by authors. Pneumonia with a fibrinous exudate is rare.

ABDOMINAL TENDERNESS. While tenderness is sometimes absent during the entire course of the disease, it is generally readily detected by the close of the first week. It may be general or localized, when the latter, it is generally on the right side of the abdomen, over the ascending colon, rather than strictly limited to the right iliac fossa, as is asserted by many authors. Too much stress has been laid upon the signs in the "right iliac fossa." I have seen many cases of typhoid fever where the focus of greatest tenderness was in the left side of the abdomen. As the intestinal glandular inflammation and ulceration is not the only factor in the production of tenderness, it will be understood why this sign is not limited to the right side, as generally believed. In certain cases of typhoid fever the glandular lesions are slight, while the catarrhal inflammation is diffuse and severe.

PAIN. Abdominal pain of a colicky nature usually precedes or attends the diarrhœal stools. Perforation of the bowel is apt to be heralded by the occurrence of sharp pains. Such pain, however, bears no relationship to the stool, is of sudden onset, constant, and attended by systemic evidences of the severity of the complications, great prostration, a variable degree of shock, nausea, vomiting, coldness of the surface, and perhaps cold sweat.

TYMPANITES. The early fulness of the abdomen gives rise in some cases in the second week to tympanitic distension, which is sometimes of high degree. After the establishment of this symptom it usually persists until convalescence. The latter condition is usually indicative of unhealed ulcers (these paralyzing the peristaltic movements of the bowels) and is therefore an important prognostic and therapeutic indication. Tympanites is generally presumed to be the result of putrefactive changes leading to ballooning of the intestines, and to degeneration of the muscular coat of the bowel. When once developed in a case it is apt to persist till the termination, though varying in degree from day to day.

GURGLING. Gurgling is found in certain cases when pressure is exerted at the right iliac fossa. Too much importance has been attached to this sign. Fluid or gas in the intestines, due to any disease or condition, is quite capable of producing this symptom, without the presence of the specific intestinal lesion of typhoid fever.

**STOOLS.** Constipation is, as a rule, present during the first week. Nearly all cases develop diarrhœa some time between the sixth and twelfth days. Exceptionally this symptom may be present from the first; it may be temporary or last throughout the course of the disease. The stools are usually of a gruel-like consistency, somewhat frothy, and of a yellowish or greenish color. The frequency and size of the stools depend more upon the catarrhal state of the intestines than upon the specific glandular lesion. They frequently contain undigested matters. If milk has been used as a food there may be innumerable minute coagula. Late in the disease the stools may become watery, dark and offensive, containing altered blood, in the form of "coffee-grounds-like" sediment, and are frequently involuntary. Exceptionally, constipation may be the condition of the bowels throughout the entire attack of fever.

**INTESTINAL HÆMORRHAGE** to some degree occurs in every fifth or sixth case and is of two kinds, capillary and arterial. The capillary hæmorrhage usually takes place comparatively early in the disease, is slight in quantity, and is apt to be dark in color on account of the longer retention of the blood within the bowel. The arterial hæmorrhage may be large or small in quantity; it may be sufficient indeed to destroy life at once. It results from the erosion of vessels involved in the intestinal lesions. Rarely is the hæmorrhage "concealed," its occurrence being known only by the discharge of dark offensive stools, which present the various appearances of altered blood. If concealed bleeding is very profuse, the various general symptoms of hæmorrhage will develop. In weakened subjects an internal hemorrhage may be sufficiently free to cause death within a short time, without the discharge of any blood *per anum*. Free hæmorrhage is frequently preceded by small quantities of altered blood in the stools. For this reason, if no other, every stool of a typhoid patient should be examined by a competent person. According to some authors small discharges of blood seem to exercise a salutary effect upon the intestinal inflammation. While this may be true of some cases, I think that the presence of blood in the stools should always be looked upon as suggestive of danger, and combated accordingly. Kraft shows that 26.2 per cent. of the cases of intestinal hæmorrhages terminate fatally; males, though less frequently affected than females, display a disproportionate mortality. A sudden falling of the temperature without apparent cause should suggest search for a concealed hæmorrhage. While profuse or frequent hæmorrhages occur as a rule in typhoids of an aggravated character, quite a percentage of cases presenting this symptom are so mild as to lead to surprise on the appearance of free bleeding. I have seen death occur upon the ninth day within a few hours of a very free bleeding in a case so mild as to make the diagnosis a matter of doubt up to that time, although a provisional diagnosis of typhoid fever had been made and the best of care bestowed.

**ERUPTION.** The eruption is highly characteristic and therefore of diagnostic importance. Some authors class it among the pathological lesions. It sometimes appears as early as the fifth day of the disease and may, in rare cases, be delayed until the twelfth or fourteenth day. It appears in small successive crops, each crop being plainly visible for about three or four days. The abdomen and lower chest are the seat of the earlier groups, although when copious, they extend to other portions of the trunk, and to the extremities. In many cases it is so scanty as to be found only with difficulty. Even the face may be involved in certain unusual instances. The evolution and decline of these eruption groups go on for some two weeks. Highly characteristic as the eruption is, it is absent in a proportion of cases variously estimated up to 30 per cent. I have found but a small number of my cases without it, surely not over 10 or 12 per cent. The spots have been aptly compared to flea-bites. They vary somewhat in size, but average from two to four millimetres in diameter. Their outlines are not sharp. They are very slightly elevated, and disappear upon pressure, reappearing immediately upon removal of the same. It is convenient to outline and date the earlier crops with an indelible pencil and thus watch their development and decline, a most important measure if one would distinguish between the eruption and the spots simulating it.

A very interesting symptom to which attention has been recently directed by Filipovich is a peculiar induration and yellowish or orange tint in the prominent portions of the plantar and palmar surfaces. The explanation of the condition is found in the enfeebled action of the heart, the incomplete filling of the capillaries, and the dryness of the skin. This observation has received confirmation from Shibnevski. It is not a newly described symptom, however, as attention was directed to it a number of years ago by the late Dr. Ellwood Wilson and it was observed very constantly in the cases treated at the Philadelphia Hospital. It was found absent in other fevers. It may possess considerable diagnostic value. It cannot yet be regarded as pathognomonic.

**URINE.** During the early stage of typhoid fever, the urine presents the general characters peculiar to the febrile state. It is reduced in quantity, of higher color and specific gravity than normal. Later in the disease the excretion is greater in quantity than normal, the amount being determined largely by the height of the fever. Although the water in the urine is increased, the relative proportion of solids is diminished; the retention of large quantities of these excrementitious matters resulting in delirium, convulsions, a gradually increasing stupor and finally coma—in a word, uræmia. Albumin is present during the latter weeks of aggravated cases, but the quantity is generally small. It usually disappears with convalescence. In a large number of cases this albuminuria is due to the action of the specific poison of the disease, and per-



haps of the retained excrementitious matter upon the impaired renal epithelium. In a few others the albuminuria is the result of an actual parenchymatous nephritis. In the latter case, casts and other evidences of renal degeneration are generally discovered. The albumin too in the latter case may persist and a chronic nephritis follow. Andre claims that albuminuria appears in the earlier stages of typhoid fever, and is therefore a valuable help to diagnosis. I have made a number of observations, discovering traces of albumin only occasionally, but its presence is not at all as constant as represented by Andre, at least in my own cases.

The greatest care must be exercised lest retention of urine take place. Neglect to look after the state of the bladder as a routine measure has repeatedly permitted this complication to occur, and this, notwithstanding the daily passage of a quantity of urine apparently up to the normal standard.

Ehrlich has recently introduced a urinary test for typhoid fever. It is made as follows: Add to twenty-five parts of a saturated solution of sulph-anilic acid in dilute hydrochloric acid 1:20, one part of sodium nitrate solution 5 per cent. in distilled water. Mix the whole with an equal bulk of urine, and render alkaline with water strongly impregnated with ammonia. After the first week and until defervescence a characteristic red color is developed. The same result, but not as uniformly, has been lately produced in the urine of persons suffering from measles, albuminous urines from cases of acute and chronic nephritis, acute general tuberculosis, chronic phthisis, and occasionally in rheumatism. Were it reliable, the lateness with which the test is applicable, *i. e.*, after the general diagnostic criteria are present, makes it of little value.

Much interest has been attached to this test ever since its announcement. Time, however, seems to show most forcibly its lack of practical value. Edwards, after instituting a number of observations, finds that it is occasionally absent in typhoid fever, and it is very frequently present in quite a variety of diseases, notably in rheumatism, nephritis, syphilis, cardiac lesions, pneumonia, plumbism, cerebral hæmorrhage, septicæmia, arthritis, neuritis, gastritis, purpura, meningitis, cirrhosis of the liver, intestinal obstruction, and intussusception. Some of these conditions, it will be seen, are ones in which differentiation from typhoid fever is often called for. As Edwards remarks, the test fails when most urgently needed for diagnosis.

**Complications, Sequelæ, and Unusual Features.**—Perforation of the bowel is a not infrequent complication of the fatal cases of typhoid fever. It is most commonly observed at the end of the second week, or beginning of the third. Exceptionally it has occurred as early as the eighth day, or as late as the sixth week, when convalescence was well advanced. In 4,680 cases of typhoid fever tabulated by Fitz, there



was a mortality of 6.58 per cent. from this cause. Pepper estimates the frequency of perforation at from 2 to 3 per cent. of all cases. Murchison states that 11 per cent. of the fatal cases are due to it. Among the Munich cases (2,000 in number) it was the cause of death in 5.7 per cent. My own experience would make this complication much less frequent than the above figures would lead one to believe, it having occurred but once in 223 personal cases, and that one of the ambulatory type. I have, however, met with it several times in cases seen with other physicians.

The perforations may vary in number all the way from one to thirty. They are generally small; at times so small as to make their detection at the autopsy a very difficult matter. In other cases they deserve the name of rents, so extensive are they.

The symptoms are nearly always sudden in their onset, and consist of acute abdominal pain associated with collapse. Hæmorrhage sometimes attends the accident. Evidences of peritonitis shortly appear, consisting of tenderness, distension of the abdomen, vomiting, and weak pulse. A valuable physical sign of perforation is disappearance of the liver dulness, as first pointed out by Austin Flint. This symptom may, however, not be available in some cases, owing to previously existing and marked tympanites. The exciting causes of perforation are the taking of indigestible food, excessive tympanites, vomiting, unusual and sudden exertion, and ascarides.

The tendency of perforation is to a fatal issue; indeed, at one time this was looked upon as the inevitable result; but recent observations show that some cases do recover. In one patient seen by me with Dr. Frank Buchman, of this city, the peritonitis subsided, and the patient died subsequently of heart-failure. An autopsy revealed a perforation in the lower portion of the ileum thoroughly repaired by plastic material. In the "walking typhoids" the symptoms of perforation and its consequences are sometimes the first indication of the serious nature of the malady. Under such circumstances the symptoms are not infrequently attributed to other than their true cause. Tympanites is so constant a symptom of perforation that we should always be suspicious if sudden distension of the abdomen supervenes, even if it be unattended by pain and other serious symptoms.

Exceptionally perforation occurs without symptoms, or with such mild phenomena as to give rise to absolutely no suspicion that such a serious accident has occurred. Under these circumstances the rupture is generally small and the resulting peritonitis of gradual development.

PERITONITIS, in the majority of cases in which it occurs, is the result of perforation; it may set in independently of that condition. Thus it may arise in consequence of rupture of abscesses of the mesenteric glands or of one of the abdominal viscera, or by extension of inflam-

mation from the ulcerations of Peyer's patches through the coats of the intestines. It may even exceptionally complicate the case independently of any other abdominal lesion.

**PNEUMONIA.** Inflammation of the lungs may develop at any period in the course of typhoid fever. It may be of either the croupous or catarrhal type. Croupous pneumonia may appear either during or at the very beginning of the typhoid fever, or it may become manifest not until the tenth or twelfth day of the disease. In the former case, all the characteristic symptoms of pneumonic fever, as ordinarily met with, are observed, the suspicion that typhoid fever is about to appear being scarcely entertained. In the latter case, the symptoms of pneumonia are by no means typical and may escape recognition.

Catarrhal pneumonia is especially apt to occur during the second half of the course of typhoid fever. It involves the lobules of the lungs by extension from the bronchial tubes. It is, perhaps, etiologically independent of the tubes in some cases. It is generally associated with hypostatic consolidation, œdema, etc. Cough and expectoration are not prominent symptoms; and if they have existed as evidences of a pre-existing bronchitis, they are likely to diminish with the establishment of the pneumonia. The symptoms of catarrhal pneumonia are not generally very pronounced. In well-marked cases the temperature averages higher than previously, and with less marked morning remissions. The circulatory symptoms are increased, respiration embarrassed, and the facial appearance often changes from the stupid one of typhoid to the anxious expression of pulmonary disorders.

Other pulmonary complications of occasional occurrence during or after typhoid fever are pulmonary embolism, abscesses and gangrene.

Pulmonary œdema and hypostatic congestion of the lungs not infrequently occur from circulatory failure and the maintenance of one decubitus for too prolonged a period.

Pleurisy very rarely becomes a complicating factor of typhoid fever. It is, however, a very serious one. The effusion may be of the ordinary serous character ending in recovery, or purulent. Sharp stitching splenic pain sometimes occurs early, and has a symptomatic resemblance to pleurisy.

**COMPLICATIONS REFERRED TO THE UPPER RESPIRATORY TRACT** are comparatively frequent. Bronchitis, which is so constantly present as to constitute a symptom of the typical typhoid fever, has already been mentioned. A catarrhal state of the nose and pharynx is more or less constant. Ulcerations of the nose and larynx are somewhat rare. The former occur late in the disease, when emaciation has progressed and vital powers are low. With the system thus depreciated, slight injuries, such as those caused by picking at the nose, may produce ulcerations of the septum, going on even to perforation.

Membranous pharyngitis is a rare but exceedingly fatal complication.

LARYNGEAL COMPLICATIONS occur with greater frequency in Europe than in this country. Gruder, who has made an extensive study of them, divides them into three classes: (1) The specific typhoid lesion which occurs only in situations where follicular structures are present, and at the same time as the intestinal ulcerations. They are thus observed on the posterior wall of the larynx, at the base of the epiglottis, and on the ary-epiglottidean folds. (2) Catarrhal manifestations with ulcerations; the ulcer may lay the cartilages bare. (3) Diphtheritic ulcerations.

It is altogether too common to look upon the hardness of hearing accompanying some cases of typhoid fever as dependent upon blunted sense-perception resulting from the effect of the typhoid poison on the sensorium. It must be borne in mind that deafness may ensue from other causes, notably from catarrhal inflammation of the middle ear, being an extension of a similar process in the pharynx, or suppuration in different portions of the auditory apparatus. The former may result disastrously to the hearing; and the latter to both hearing and life. It is true that these aural complications are not of frequent occurrence; but it is this very infrequency that leads to their neglect and proper management. But little if any attention has been paid to this subject by competent authorities, so that the only review of "the disorders of the ears in typhoid fever" with which I am acquainted (*Medical Record*, Sept. 3, 1892), is attended with unusual interest. The treatment of such a complication of course differs in no wise from that useful in similar pathological conditions occurring as primary disorders.

THE CIRCULATORY COMPLICATIONS of typhoid fever have in great measure been described under the circulatory symptoms of the disease (see page 55). It remains only to refer to the possible, infrequent though it be, occurrence of endo- and pericarditis and myocarditis, among cardiac affections; and arteritis and embolism among accidents to which the peripheral circulation is liable. Gangrene of an entire or of a portion of a limb may thus occur, and must be regarded as a very serious matter. Venous thrombosis offers a favorable prognosis in the majority of cases. The affected limb, however, rarely regains its former size.

Quite a variety of suppurative conditions may occur during the late stages of or convalescence from typhoid fever. Hardly a tissue or organ of the body is exempt. Numerous subcutaneous abscesses, suppuration in a goitrous thyroid gland, abscess of the spleen, liver, lungs, kidneys, and other viscera, inter- and intra-muscular suppuration, suppurative epididymitis, noma, parotitis, suppurative periostitis, especially of the ribs and tibia, etc., have all been reported. In most cases bacteriological examination of the pus reveals the presence of the typhoid bacilli; in the minority of cases, the bacilli of suppuration only are found.



The periosteal complications sometimes take unusual forms, as witnessed in a case reported by Eskridge, in which periostitis affected the pelvic bones and was associated with pachymeningitis.

Suppurative parotitis is an especially fatal complication. It is usually unilateral.

ALBUMINURIA is occasionally present in typhoid fever. It may be but a simple febrile albuminuria or dependent upon an acute nephritis. Following convalescence, it is generally the result of a post-typhoid pyelitis.

As to the condition of the general health after typhoid fever, much has been said among the laity to the effect that patients improve wonderfully. This is true in a measure only. In the majority of cases the patient regains his wonted condition. In a minority—and it is an equal chance which of these will follow—unusual increase or depreciation of the patient's health standard ensues.

In the majority of cases of typhoid fever patients, the hair falls out for some time after convalescence has been established. This symptom causes women considerable concern. As a rule this alopecia is but temporary.

HERPES LABIALIS is seldom present. Some form of paralysis may develop during, but more frequently after the fever. It is usually dependent upon a neuritis. Recovery ultimately occurs in most instances. Epilepsy, transitory aphasia, slow articulation, mental aberration of various types, defective memory, tremor or choreic movements are rare results. The nervous symptoms of this disease are in general due to disturbed nutrition and the prognosis is favorable. Obstructive jaundice, hepatic abscess, and acute yellow atrophy of the liver are infrequent. Catarrh of the bladder is generally the result of a want of attention to thorough antiseptic precautions during catheterization. Pyelitis of an ulcerative or pseudo-membranous character may occur. Typhoid fever may be complicated by other infectious diseases, such as scarlatina, erysipelas, influenza, smallpox, etc. Gastric ulcer may lead to free vomiting of blood, and paretic pharyngeal muscles to dysphagia. Epistaxis or a free flow of blood from the posterior nares is not unusual. The nails often possess transverse ridges, due to the impairment of their nutrition, and the skin remains dry and ill nourished for some time.

RELAPSES. It has become fashionable to speak of sudden elevations of temperature without aggravation of symptoms appearing at the apparent close of the case, and which continue for but a short time, as recrudescences. The term relapse is best reserved to designate a second attack of typhoid fever following immediately on the first, and supposed to be due to re-infection, perhaps auto-infection. The former may result from a variety of complicating conditions, in turn excited by imprudences in diet, exercise, etc.; but a relapse as the term is now used indicates a fresh attack of the disease, presenting the usual symptoms, general course,



and pathological lesions of the primary disease. Post-mortem examinations of persons dying of such relapses show both the unhealed ulcers of the primary and the less advanced changes of the secondary attack.

**Varieties.**—**WALKING CASES.** In certain mild cases the symptoms are so slight that the patient keeps about for one or two weeks. The clinical course of the disease in such instances is usually regular, but the symptoms are all mild in degree. The diagnosis under such circumstances is often difficult, as the eruption is usually scanty, the enlargement of the spleen not sufficient to be easily detected, and if diarrhœa is present, the stools are infrequent, and often afford a sense of relief. Many remain constipated during the entire course of the attack. Notwithstanding the mildness of the symptoms, the essential lesions may progress rapidly to the extent of perforation of the bowels; symptoms of collapse and peritonitis being the first to attract attention to the serious nature of the case.

**ABORTIVE CASES.** Certain cases of typhoid fever follow a regular course up to about the fourteenth or fifteenth day, when defervescence occurs. These are known as abortive typhoids. They may start out very violently, and give every indication that the disease will be continued to its usual, if not to a greater, length. The subsidence of the temperature to a normal point and convalescence is very rapid. For a long time cases of the abortive type have been considered doubtful, the opinion being general that typhoid must of necessity pursue a typical course. But aside from general diagnostic evidences, there can be no doubt that such typical cases do exist, a sufficient number of post-mortem examinations having been made to demonstrate the fact, death having resulted from indiscretions in diet, etc., after the subsidence of the fever and apparent convalescence of the patient. Reasoning by analogy, we can see no reason why abortions of typhoid fever may not occur at even much earlier dates, though we are not always able, with our present knowledge, to diagnosticate the disease with certainty during the first week, and for this reason the subject must remain in doubt. The absurd statements so frequently heard to the effect that typhoid fever has been prevented or broken during the early days of the attack, are not warranted, and indeed are not made by physicians who are competent to speak upon the subject. While such may be the truth, it is a supposition or bald statement only, without logical support.

There is another form of typhoid which may be called abortive, and of this I am enabled to present the accompanying temperature chart (see Figure 4) through the courtesy of my colleague, Dr. Haines. In this case the temperature fell to normal as indicated, and remained so throughout the further progress of the case. At the same time all the other characteristic symptoms of the disease continued throughout, and the case ran what is ordinarily called a normal course.

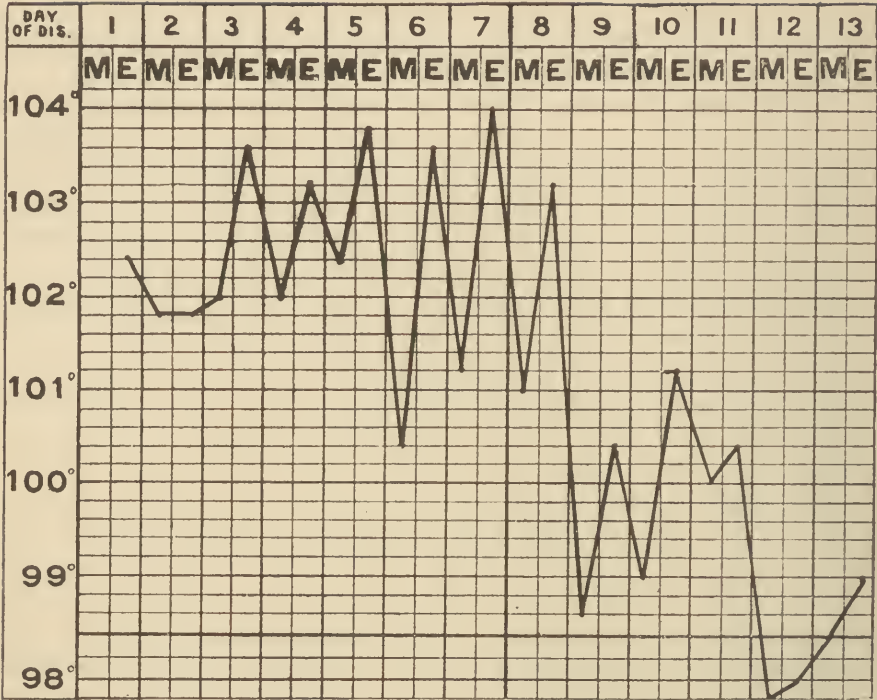


FIG. 4.—TEMPERATURE CHART OF AN ABORTIVE CASE OF TYPHOID FEVER.  
FROM THE HAHNEMANN HOSPITAL.

Cases in which the nervous symptoms are early and continuously prominent, cases in which the brunt of the disease seems to be borne by the nervous system, are frequently termed "*cerebral*" or "*nervous*" typhoid. If the intestinal lesion and its attending phenomena are the most prominent—*abdominal typhoid* ; if lung conditions attract the most attention—*pulmonary typhoid*. Such a nomenclature possesses the merit of conciseness.

**Diagnosis.**—In considering the diagnosis of typhoid fever my statements will have reference especially to the first eight days of an attack, or until the appearance of the eruption, which clears up all doubt. The suggestive data are the prevalence of the disease in the neighborhood, a prodromic period of indefinite complaints, which is, however, often absent, and a continued fever which does not seem to be related to any local lesions. While the temperature rise is often characteristic, it may be sudden in its elevation or intermitting during the early days. This fact I pointed out in a paper read before the Philadelphia County Homœopathic Medical Society, and in which I dwelt especially on the great variety presented by the invasion symptoms. Jaccoud has recently called attention to the same feature of the disease. Towards the close of

the first week there may be noted enlargement of the spleen, the gradual development of abdominal symptoms, one to two degrees elevation in the surface temperature over the right iliac fossa and spleen, and sometimes over the kidneys, and finally the eruption which may be present as early as the fifth day, although usually not before the seventh or eighth day. Ehrlich's urinary test may corroborate the diagnosis by the last of the week, still, as already stated, its value is limited because of the positive character of the symptoms by the time it has developed.

As the diagnosis must always remain in some doubt prior to the appearance of the eruption, and as the first week is the most important period of the disease therapeutically, it is necessary to impress the necessity, if we expect a favorable result, of considering all continued fevers as typhoids until the progress of the disease demonstrates the contrary. If found to be in error, the mistake does not bear evil fruit. The great predominance of typhoid fever over all other continued fevers in this and most countries, and the necessity of getting the disease within control in its formative stage, is, we think, sufficient reason for advising such a radical position.

In children and aged people the course of the disease is frequently irregular, leading consequently to increased difficulties in diagnosis.

Pneumonia, pyæmia, septicæmia, acute tuberculosis, gastro-enteritis, etc., are the diseases which are most frequently confounded with typhoid fever.

Typhoid fever, which in its early stages manifests prominent pulmonary symptoms, is easily mistaken for pneumonia. The pneumonia which complicates typhoid fever is stated to generally come on late in the disease, but we have met several notable exceptions to this statement. Two cases have been admitted to the Hahnemann Hospital within the past eighteen months which were supposed to be suffering from primary pneumonia, until further development revealed them to be examples of typhoid fever. The abrupt beginning of some typhoids, —the presence of chill, rapid rise of temperature, cough and pain in the chest with signs of consolidation, are apt to lead the mind away from that disease.

Pneumonia of the aged sometimes presents a striking general resemblance to typhoid fever. The slight cough, the frequent absence of chill, pain, and expectoration, the high development of the typhoid state, all tend to mislead. The history of the case, the absence of eruption, enlarged spleen and increased heat over the right iliac fossa and spleen, point to the pneumonia as of primary occurrence.

The resemblances existing between typhoid fever and acute phthisis or general tuberculosis, are sometimes striking, baffling for the time the most experienced examiners. The latter affection has a remitting fever of high grade, high temperature is reached early, and averages higher



than in typhoid fever, being frequently  $106^{\circ}$  F. and higher, while typhoid fever rarely attains such a high point before late in the second or third week. Again, the location of the consolidated area in typhoid fever is at the base of the lung; in phthisis it is generally at the apex. In acute tuberculosis there are general bronchial râles, mainly subcrepitant, with but slight general impairment of resonance, unless catarrhal phthisis pre-existed, when there will be circumscribed areas of consolidation. Bouchut claims that the ophthalmoscope reveals tubercular granules in the choroid in all cases. We think that there are exceptions to this statement, as eminent ophthalmoscopists have verified my failure to discover them in a number of instances. The presence of delirium, a dry brownish tongue, sordes on the teeth, mental hebetude or decided stupor, progressive emaciation, and loss of strength, and abdominal and bowel symptoms, similar to typhoid fever, make up a strong resemblance. After the first week the appearance of the eruption clears up the diagnostic fog.

Gastro-enteric inflammation in children is sometimes indistinguishable from typhoid fever. This is largely due to the very atypical course of infantile typhoid fever. In them the typhoid state is seldom marked, the eruption is often absent, the temperature irregular and subject to unaccountable fluctuations, though rarely higher than  $103.5^{\circ}$  F. and usually under  $103^{\circ}$  F. The history assists; many of the symptoms in gastro-enteric inflammation precede the fever. The gastric disturbance, the diarrhœa, tympanites, etc., increase the similarity. When occurring in adults the differentiation of these conditions is usually an easy matter.

**Prognosis.**—A very important point, and one which greatly assists in the framing of a prognosis, is the determination of the question as to whether or not the patient was going about during the first week of the fever, such action seriously jeopardizing recovery.

A sound prognosis can only be rendered in any case of typhoid fever after a thorough consideration of all the important features of the case. In no disease is such a broad view of the patient's condition more essential to prognostic indications. Leaving out of consideration, for the present, so-called complications (many of which are simply aggravated states of what we may call normal features in the course of the disease), it may be stated that the temperature chart, carefully kept, will afford the most unerring information. The height of the temperature, the duration of the diurnal aggravation, the length of the febrile state, the temperature indications about the close of the first and second weeks, all give most valuable information as to the future of the patient. It is seldom that one must prognosticate, as to the future danger to the life of the patient, before the second week. If by the eighth or ninth day the temperature has reached  $105^{\circ}$  or more, and has attained this point



by what may be designated a gradual rise, the physician is warranted in indicating a stormy second week; the temperature will probably remain continuously as high or even ascend, and correspondingly severe symptoms will attend. If the rise to this high point is rather sudden, complications should be searched for with great care. Such comparatively sudden rises may be temporary only; but oftener, even if due to a complication, the essential fever is aggravated and the temperature remains high, even though the complication be removed. This I have witnessed in connection with the supervention of respiratory and intestinal conditions. In the second week much can be learned by watching the daily temperature aggravations and ameliorations. If the fall, which usually occurs after midnight, is followed by a gradual morning and forenoon rise—a long remission—if the evening rise is a little less each day, it augurs well for the future intensity and duration of the attack. Sudden elevations and depressions at any period of the disease should excite alarm, as they indicate a complication of some sort. Rapidly falling temperature suggests concealed hæmorrhage.

Next in importance to the temperature, as a prognostic element, is the heart—an organ which should, from the first day of the physician's attendance, claim his careful attention. Many persons, especially those advanced in life, enter upon the course of typhoid fever with hearts already damaged, hearts which, in the best health the patient enjoys, perform their functions but feebly. Such organs are liable to soon give up the struggle under the increased stress induced by continued fever. If the physician can discern this condition early, it leads to valuable prognostic and therapeutic results.

Attention to the pulse and auscultation of the heart afford the most information. Frequency alone is of far less significance than impaired force and disturbed rhythm. There are few conditions which warrant a bad prognosis if the pulse is regular and strong.

**Treatment.**—GENERAL MANAGEMENT. Attention to the arrangement of a suitable room and to the personal hygiene of the patient, are the subjects claiming first attention in the treatment of typhoid fever. A large, well-ventilated quiet room should be selected. The carpet and all unnecessary furniture and hangings should be removed. Especially should upholstered furniture be taken away. To lessen the noise incident to walking about the room, mats should be laid. Thorough ventilation and a subdued light must be maintained throughout the twenty-four hours, and if the season permits of it, the thermometer should register about 65° F. A nurse who has had experience in the management of typhoid fever patients is desirable. All important instructions of the physician, and all reports of the nurse to the physician, should be in writing. Especially should records of the temperature, respiration, pulse, stools, food, etc., be accurately kept, and furnished the physician at each

visit. The bed-linen as well as the clothing of the patient should be changed daily. This direction is an imperative one if diarrhœa be present. The body should be sponged over its entire surface once or twice daily, using for this purpose water as cool as can be employed without exciting shivering or unpleasant sense of cold. For the earlier sponging tepid water can be advantageously employed, and a change to cooler water made gradually. The mouth must be kept scrupulously clean ; it should be washed twice daily. Sordes should be removed with cotton wound on a small pair of forceps or on a soft pine stick. It may be necessary to soften the deposit with glycerine before proceeding to its removal. Neglect to properly cleanse the mouth may lead to parotitis and otitis media. Cleansing with hot water and soap should never be neglected. Unfortunately, this precaution is not observed by many otherwise good nurses. All but the attendant should be banished from the sick-room, and all duties about the room should be performed quietly and methodically.

The position of the patient should be changed several times during the twenty-four hours, as the maintenance of one decubitus for too prolonged a period favors gravistatic pulmonary congestion, the formation of bedsores, etc. The importance of putting the patient suspected of having typhoid fever to bed early, cannot be emphasized too strongly. This rule we find violated almost constantly, often indeed by physicians of large experience. As a diagnosis cannot be made with certainty the first week, we can only do our duty by our patients, if, as I have already suggested, we call all continued fevers typhoids until evidence to the contrary is abundant. Economy of the patient's strength must be practised from the early days of the disease. To secure this, interdict sitting up, even in bed, all unnecessary conversation, movements and company ; even rising to stool or urine is to be positively prohibited. The bed-pan, not the commode, must always be used. After the disease has fully developed, the necessity for these precautions becomes still greater, especially on account of the impairment of the heart's power. The frequent changes of position necessary at this time must be accomplished as gently as possible, and preferably by manipulations with the under sheet. The patient must be disturbed as infrequently as proper attention will allow. In sponging, for instance, treat only that portion of the body which can be reached at the time, completing the bath after the next change of position. The ease of the nurse is not to be considered if it detracts in the slightest degree from the proper carrying out of the most rigid care. It is probable that under the Brand system of cold-water treatment such extreme care is not necessary, although the condition of the patient should decide this point.

As the typhoid poison is excreted mainly in the stools, the necessity for their thorough disinfection with some substance known to have the

power of destroying the poison, is apparent. Many methods of disinfection of the excreta have been advised, some valuable, but many worthless. One which is nearly always and everywhere available is that with chloride of lime. A good reliable preparation of this chemical must be secured. It should be made into solution, in the proportion of six ounces of chloride of lime to one gallon of water. Of this from one pint to one quart should be used for the treatment of each stool or quantity of vomited matter. After remaining in contact with the disinfecting fluid for an hour, the stools may be thrown into the water-closet. This is far better than burying them, for that leads to ground soakage.

DIET. The articles of diet must be of the most nutritious kind, invariably liquid, and administered in small, measured quantities, and frequently. Sterilized milk is undoubtedly the most valuable, and one which agrees well with most patients. There is, however, always a small number of patients with whom milk does not agree, and frequently this disagreement can only be discovered by a careful inspection of the patient's stools. I desire to emphasize these statements for the reason that early teachings, to the effect that milk is *the* food for typhoid fever and should be administered to every case and rarely disagrees, have been generally accepted with serious results to many patients. Milk must be stopped entirely, or prepared in some manner calculated to increase its digestibility and persisted in most cautiously if any number of coagula appear in the stools. The amount given at one time, and the frequency of repetition depend upon the age, appetite and digestive ability of the patient, as well as upon the amount of other nutriment the patient may be taking. When milk is the sole article of diet, from two to four pints should be given in the twenty-four hours. The latter quantity, I think, is as large as should ever be given. Milk is often objected to upon the ground that the patient does not like it or has never been able to digest it. The latter statement is usually without foundation, and if a milk diet is firmly insisted upon, it will generally be taken and without ill effects. If milk produces constipation this result may be often overcome by peptonizing it, adding lime or plain soda water, giving all or a portion in the form of koumiss, matzoon, buttermilk, junket or cottage cheese. Skimmed milk will sometimes agree with the stomach better than the unskimmed article. Gelatin is a valuable article of diet, and a little may be administered daily with advantage. It is not contraindicated by any condition of the patient. The question of overfeeding has been discussed by the author in a paper read before the American Institute of Homœopathy in 1888.

In that paper he said: "Disturbances due to the former (the influence of the typhoid poison on the nervous system) are more important, for depressed function resulting from central nervous changes is always difficult to deal with. During this period alterations in the epithelia,



glands and muscles are generally very slight, and disturbed digestion is due more to impaired nervous power than to changes in the anatomical elements of the stomach.

"In the second week the toxæmia, with its nervous results, continues; but structural alterations of the elements are now becoming more marked. The cell struggling under the depressing influence of the typhoid poison is but imperfectly appropriating and transforming the impaired pabulum which is offered it. Consequently much that it takes remains within the cell imperfectly assimilated and, therefore, a burden. Excretion of its products is correspondingly imperfect. It is in this stage of the disease, and later, when the structural alterations are profound and prostration (*adynamia*) is excessive, that we are advised to pour in the food. Two to three quarts of raw milk, one to two pints of beef tea, and perhaps a little farinaceous gruel with (often) six to twelve ounces of whiskey or brandy, are frequently given within the twenty-four hours.

"Add to these conditions of the digestive organs and nervous system the blood struggling under the oppressive load of imperfectly elaborated material, the specific poison causing the fever, and the largely increased amount of irritative excrementitious matter—add to these the crippled glandular organs, imperfectly elaborating and depurating; the general tissue elements, their integrity impaired—and you have some of the reasons why the processes of life should not be still further disturbed by the administration of quantities of food, often larger than the normal system can properly dispose of."

When diarrhœa is obstinate the problem of proper diet becomes for a time a most important one. We can recommend the giving of one ounce of whey mixed with one drachm of burned brandy each hour. Every three or four hours, two ounces of carefully made beef tea may be given. This may be peptonized in some cases. Personally, I do not share in the current opposition to this food. In some cases a thin peptonized farinaceous gruel, as arrowroot, cornstarch, etc., is well borne. Following suggestions growing out of recent experimentation, we cook such gruels for several hours, thus increasing their digestibility. In occasional cases, certain of the prepared cereal foods and meat extracts agree well. Plain (non-fruit) ice-creams are sometimes serviceable. With convalescence the variety in diet is gradually increased, and solid food of a non-fibrous sort added about one week after the temperature becomes normal, provided diarrhœa, distended abdomen, etc., have entirely disappeared. Meat should not be permitted for from two to three weeks after convalescence has been established. Fruits which contain seeds should not be permitted at even this late date.

STIMULANTS are invaluable when indicated. They should never be administered as a routine measure. After prescribing them, their action



should be carefully watched to determine whether or not they are accomplishing the purpose for which they are employed. For indications both patient and symptoms are to be considered. Murchison's rule respecting the patient is worthy of consideration. He says that patients under twenty years of age, as a rule, do better without alcohol, while most patients beyond the age of forty may take it with advantage after the first week of the fever. The previous habits of the patient must also be considered. Those who have been habituated to alcoholic indulgence require stimulation earlier and more liberally than do others.

The symptomatic indications for stimulants are found in the heart, the tongue and the brain. If the heart's action becomes weak and frequent, the pulse irregular, the tongue brown and dry at its edges, and delirium appears, whiskey or brandy is indicated. Burgundy, port, claret or champagne may also be administered, if more agreeable to the patient's palate. When albuminuria is present, alcoholic stimulants must be used with caution.

As to dose and administration, these must be varied according to indications. From four drachms to two or more ounces of whiskey or brandy may be given every three hours. If Burgundy, claret or champagne be used, as much as one and a half pints may be given in the twenty-four hours. As a rule more stimulation is required during the night and early morning hours. Repetition of dose should be governed entirely by the effects produced.

DRINKS. Pure water should constitute the principal drinking fluid. If there is the slightest suspicion respecting the purity of the water employed by the patient or his family, it should be boiled. Drink should be offered frequently, even if not requested, as in conditions of mental hebetude, the patient's indifference may prevent him from making his wants known. For variety's sake, coffee, tea, wine, or any alcoholic may be added in small quantities to the water. If the abdomen is not distended, carbonated waters are grateful and are not objectionable. They may be flavored with any of the above. Fruit-syrups are objectionable. The addition of simple syrup is generally detrimental, as it provokes flatulency.

The drinking of large quantities of water was erected into a special form of treatment by Debove a number of years ago. He made his patients partake freely of water, even to the extent of five or six quarts daily. Lichtheim and Maillart have followed this plan in a number of cases with excellent results. Fever, dryness of the mouth, and the nervous symptoms disappear or are controlled. I am satisfied that the free administration of water is of great value.

As the continued administration of milk, which is both food and drink, is likely to become very objectionable to the patient's palate,

coffee, tea, wine, brandy or whiskey, liquid or dry malt extracts or plain soda water may be added according to the judgment of the physician. I have several times allowed beer, and seemingly with advantage. Beer exercises stimulating, digestive and sedative influences.

Lemonade and other acid fruit-juices are not allowable.

After the physician has passed in review all the features of the case, and considered all questions of general management, he is then, and then only, prepared to deliberate upon the medicines to be prescribed. Unfortunately the order just indicated is too often reversed in practice. Medication can be of little service while the influence of diet, undue exertion, etc., are practically ignored. We are to accomplish with medicines only what we are unable to secure by other means. During the early stage the diagnosis is usually in some doubt; but the possibility of typhoid should never be lost sight of in any case of continued fever. The patient must be thoroughly examined, and a medicine selected which corresponds most nearly with the true totality.

**MEDICINAL TREATMENT.** During the first week, unless there are clear indications for another medicine, it is my custom to administer *bryonia* from 1x to the 3x dilution, twenty drops to four ounces of water, the solution to be given in teaspoonful doses every one to three hours as required by the urgency of the symptoms. Clinical evidence of the pre-eminent value of *bryonia* at this period is overwhelming. This remedy should be continued as long as the progress of the case is satisfactory, regardless of the presence or absence of diarrhœa.

*Baptisia*, administered in the same manner as the preceding drug, is preferable if a bad state of the mucous membrane is apparent. It is suggested by dry mouth, heavily coated tongue, showing the imprints of the teeth, offensive breath, anorexia, nausea, tender epigastrium, abdominal flatulency, and perhaps diarrhœa with offensive stools. The pulse is soft and lacks tone. A little later the peculiar delirium of this remedy may be present and is a valuable indication. Some care is required to avoid giving this remedy when *mercurius* is indicated. As an "essential" typhoid remedy *baptisia* is not comparable with *bryonia*. The confidence in its abortive influence over typhoid fever which many physicians entertain, has not been warranted by my experience.

*Gelsemium* is to be preferred when prodromes consisting of a marked degree of prostration, chilliness, loss of appetite and pains of a migratory, "neuralgic" character are present from the start. The tongue is clean, the mind clear, but the patient may complain of severe headache, especially in the occiput and extending forward. These patients are often relieved by small doses of alcoholics.

*Pulsatilla* has afforded help in cases in which the gastric symptoms peculiar to this medicine were prominent. The fever is slight, the pulse soft and quiet, the patient loathes everything, even water, and the tongue

is probably thickly furred. It is indicated especially in women who suffer from menstrual derangements suggesting the medicine.

The headache of the early stage is often relieved by *belladonna*. So annoying is this symptom that one must often interrupt the action of the more essentially anti-typhoid medicine in order to give the patient relief. If the headache is frontal, and there is sensitiveness to motion, noise, or light, a few doses of five drops each of the third decimal dilution of *belladonna* may relieve. So severe may this symptom become in occasional cases that one must bear in mind special remedies for it, generally *spigelia* 2x, *antipyrin* 1x, and in rare instances, *morphia*.

The general pains in the back and limbs often lead to great restlessness and distress. *Rhus tox.* will most frequently be found indicated and successful in mitigating the pain. Headache and sleeplessness, if present, are often relieved at the same time. *Rhus* is seldom called for during the first week, excepting for the control of these symptoms.

*Antipyrin*, in doses of three grains of the first decimal trituration hourly, has helped when *rhus* has failed.

Rarely, the recurring epistaxis requires control, and has always, in my experience, disappeared after the use of *belladonna*, *hamamelis*, *crocus*, or *ipecac*.

With the advent of the second week, with its intestinal ulceration and development of the typhoid state, another class of medicines must be studied. Here testimony is almost a unit in favor of *rhus toxicodendron*. The symptoms of this stage are found wonderfully well pictured in the provings of this remedy. A dry brown tongue or red-edged irritable tongue, lips dry and brown, the teeth covered with sordes, a besotted appearance of the countenance, restlessness and weak pulse, distended and tender abdomen, and not very marked diarrhœa are some of the symptoms suggesting its use. *Rhus* is best administered in the third decimal dilution, twenty drops to four ounces of water, and teaspoonful doses given every one to four hours.

At this stage we frequently meet cases with catarrhal enteritis as the most prominent condition. The abdomen is distended, there are gurgling and pain in the bowels, especially before stools which are gruel-like in consistence, frothy, yellowish or greenish in color, and ranging in frequency from three or four to fifteen or twenty daily. The tongue may be moist; the mind is somewhat dull, and the patient quiet. For this condition *phosphoric acid* 3x dilution, twenty drops in four ounces of water, and given in teaspoonful doses every one to three hours, is at times highly effective. Apathetic cases without much brownness and dryness of the tongue often respond to *phosphoric acid*. The tendency to pass into a quiet stupor without previous mild delirium exists in a small percentage of the cases. Besides *phosphoric acid*, *arnica*, *lachesis*, *opium* and *nux moschata* should be compared for this condition.



If symptoms of great irritability of the mucous tracts appear, and *rhus toxicodendron* does not control them, if the tongue is red, raw, cracked, or glazed, if food is badly borne, thirst marked, abdomen tympanitic and tender, the stools offensive and watery, and containing decomposed shreds, three medicines suggest themselves, namely: *Cantharis*, *Lachesis*, and *turpentine*. *Cantharis* is my first choice. The indications for its use are strengthened by the presence of symptoms of irritation of the urinary tract. Ten drops of the first decimal dilution should be dissolved in two ounces of water, and one teaspoonful given every two hours.

*Lachesis* is preferable if there is marked somnolence or a comatose state. I use the 6x, ten drops in two ounces of water, one teaspoonful every two hours.

*Turpentine* in doses of one minim every hour, if the tongue and abdominal symptoms (tympantites, etc.) are prominent.

In cases that develop a heavily coated tongue with offensive breath, nausea, perhaps vomiting, uneasiness and tenderness at the epigastrium, slight jaundice, constipation, and the minimum of nervous symptoms, *hydrastis*, one drop every one to two hours, is often useful. *Hydrastis* has a decided influence upon the fever and can safely be continued after the immediate relief obtained from its employment. *Baptisia* is sometimes useful for similar symptoms, but is less often valuable.

The past few years have enabled us to add to our armamentarium valuable remedies for the treatment of the nervous symptoms of typhoid fever; *acetanilid*, *agaricus* (*agaracine*), the long employed *hyoscyamus*, and the recent but more valuable *hyoscine hydrobromate*, have enabled us to better control the more violent forms of delirium than heretofore. The mild cases seldom require special treatment; but the nervous symptoms are often of such serious import as to demand control at all hazards. Many a furious maniacal delirium is unfortunately permitted to continue until the patient is exhausted by physical fatigue and loss of sleep.

*Hyoscyamus* has been the most frequently indicated as well as the most reliable remedy in the past. Innumerable symptoms are added to the indications for the medicine as detailed in many works. "For a furious delirium, if *belladonna* has no effect, it is pre-eminently indicated in every stage, also if the patient is sunk in a state of apathetic stupefaction" (Jahr). A symptom often observed, and one which has frequently been verified personally, is "gushes of sweat" (*agaricine*).

When the patient is in a frenzy of excitement, sleepless, face haggard, pulse failing, *hyoscine hydrobromate* 3x trituration, one grain every hour, seldom fails within six or eight hours to bring quiet and a refreshing sleep. The remedy should not be discontinued as soon as relief is secured, but continued at from four to six hour intervals as long as the course of the case is favorable, as it exercises a most favorable influence upon the fever and the general disease.



*Stramonium* may succeed if *hyoscyamus* fails, especially if the delirium is of a cheerful kind, singing, whistling, mind very active, body also, automatic motions with arms, etc., especially if frightful phantasms seem to start out of the ground at the patient's side. (Jahr.)

*Agaricus* has been recommended by Dr. G. C. Hibbard for the delirium of typhoid. But Dr. Seth Pancoast long since made the same suggestion to me. While not as certain to bring at least temporary benefit as *hyoscine*, its influence is very general. Dr. Pancoast recommended ten drops of the tincture in two ounces of water, teaspoonful doses hourly, and I have followed his advice with good results. Recently I have made use of *agaricine* 1x trituration, one grain every one to three hours, and with even more than satisfactory results. The special symptoms calling for *agaricus* are: constant delirium, tremulous propulsion of the tongue, trembling of the whole body (Hibbard); typhoid fever in drunkards (Chargé); to which I would add, rigidity of the limbs, delirium with utter want of comprehension of questions; imbecile look.

*Arsenicum* has been the most disappointing medicine I have prescribed in typhoid fever. Perhaps I defer its use until the full arsenic picture (a most unpromising one) has fully developed. Jahr's advice to give it after *rhux tox.* has seemed to me valuable. The picture is one of utter prostration, to which are added the symptoms developed by extensive sloughing lesions in the intestines, heart-failure, and anæmia of the brain.

*Hydrochloric* acid and the closely related mineral acids are called for when the muscular system endures the brunt of the attack. Muscular degeneration and its consequent paresis lead to such symptoms as sinking towards the foot of the bed, patient quiet, helpless, audible descent into the digestive tract of liquids swallowed, weak, irregular intermitting pulse, mental operation repeated slowly. These are desperate cases, but *hydrochloric acid*, 1x dilution, ten drops in two ounces of water, in doses of a teaspoonful hourly, sometimes helps.

Hæmorrhages from the bowels, in a small percentage of cases, are sufficiently profuse or frequent to require energetic treatment for a time at least. Slight loss of blood may be apparently beneficial to the patient, or, at least, be attended with no untoward results. But the treatment of this symptom should not be neglected because of this fact. The appearance of the blood in the stools should stimulate the medical attendant to increased care. Rest, complete rest, proper food, *i. e.*, a food which is followed by smooth, well-digested stools, should be secured at once. If seemingly well-indicated remedies have not prevented the bleeding, or controlled it, a medicine must be selected with especial reference to this symptom. Of these, *hamamelis*, *terebinthina*, *hydrastis*, *cinchona*, *ipëcacuana*, *ergot*, and *morphia* should be especially considered.

*Hamamelis virginica* has seemed to control the bleeding in several

cases, twice being indicated by a bruised, sore sensation in the lower portion of the abdomen. One patient received five drops of the third decimal dilution hourly; the second, the tincture, twenty drops in four ounces of water, of which solution teaspoonful doses were given hourly.

*Terebinthina* is a far more important remedy in the treatment of hæmorrhages of every character than is generally appreciated. In intestinal hæmorrhage it is suggested by meteorism, tenderness of the abdomen, flatulency, offensive stools, dry mouth, brown tongue, and, possibly, urinary symptoms. It is strongly indicated if albuminuria exists. The lower dilutions and sometimes drop doses of the pure turpentine are suggested.

*Hydrastis* is indicated in patients who have heavily coated tongues and many symptoms which practitioners generally speak of as "bilious." Drop doses of the tincture have given good results.

*Ipecac* is called for when there is sharp colic below the navel. The gripings are described as if "of talons."

The profuseness of the hæmorrhage, or its frequent repetition, sometimes calls for immediate relief. I have seen two young men with mild typhoid symptoms die within a few hours from a single hæmorrhage. If the loss continues despite the best selection which can be made, we must administer hypodermatic injections of *sulphate of morphia*, using from one-eighth to one-quarter of a grain, and repeating the dose within a limited time if necessary. It is safer to give one-quarter of a grain to an adult if there are no contraindications. If resorted to, the patient should be kept under the influence of the morphia until danger of recurrence of the hæmorrhage has ceased. This demand for morphia must necessarily be rare, and must be regarded as a surgical remedy—a splint for the bowel. Alcoholics act well with the morphia. The ice-bag is strongly advocated by some; but I have not been able to satisfy myself of its value, excepting in cases of hæmorrhages associated with hyperpyrexia. The bag should be large enough to cover the entire abdomen, the latter being protected by layers of flannel.

The hypodermatic injection of *ergotine* in doses of three to ten grains is fashionable, but disappointing in its results.

Perforation of the bowel should be treated according to the principles and indications given under peritonitis.

Tympanites is to be controlled by care in diet; cold to the abdomen, and a carefully selected remedy, having reference especially to the abdominal symptoms. *Rhus*, *arsenicum*, *turpentine*, *phosphoric acid*, and many other medicines show influence over this condition.

The *arsenite of copper* has of late proven of great value for the control of the gastro-intestinal symptoms of typhoid fever. Frequent painful movements are often quickly checked and the general condition of the patient much improved. This medicine has been extensively used

recently by some observers as an essential remedy, and with excellent results.

If the accumulation of gas is in the intestinal canal, the thorough use of the rectal tube is advisable. If gas passes upon the introduction of the catheter into the rectum, but the distension is not relieved, a long rectal tube may be used. Such a tube can be passed high into the bowel by injecting a little water when the point of the tube is arrested by the folds of the gut. If the accumulation of gas is in the peritoneal cavity, and relief is demanded, the aspirator must be employed.

FEVER. Since the introduction of the clinical thermometer the importance of a high temperature has undoubtedly been exaggerated. The recognition of this fact is leading, I think, to a depreciation of its importance. The pendulum has now swung both ways, and we may hope soon to arrive at a true estimate of the subject. In typhoid, as in other fevers, we must judge of the importance of the elevated temperature in a given case by the character of the associated symptoms. The most important guides are the condition of the nervous system and the heart. Delirium, which is increasing; stupor, which is deepening; heart feebleness, which is becoming "heart-failure," suggest the importance of reducing the temperature. This must not be accomplished hurriedly, or by the use of medicinal antipyretics, before simpler measures are tested. First review the case carefully in its every feature, both in reference to its general care and the medicine prescribed. If assured that the best has been done, and the result is unfavorable, apply cold water to the surface with a sponge or by pouring (the patient being upon a rubber sheet covered with a woolen blanket). The temperature of the water should be about 90° F. upon commencing, but may be gradually cooled to 70° F., or less, according to its influence. This should be tested by the thermometer in the rectum. An important feature is the exposure of the cutaneous surface of the patient to the atmosphere during the bath, which should last twenty minutes. Properly carried out, this method usually reduces the temperature about two degrees, which is often all that is required; indeed, it seems more rational to simply reduce the heat to a thoroughly safe temperature than 3° F. to 5° F., as is often done. Before and after the bath a little stimulant had better be given. With a large rubber sheet and a tub to catch the water used, almost as effectual bathing can be carried out in this manner as by means of the tub.

The use of the bath in the treatment of continued fevers was introduced by Currie of England during the latter portion of the last century, but was little employed until forced upon our attention by Brand and his German colleagues. During the past decade this method has gained a firm foothold in America, and at present many hospitals treat all suitable patients in this manner. The best results are secured in those who



have not yet passed the fifth or sixth day of the disease. Brand and his followers claim that almost every case treated thus early will recover. After the treatment is instituted, only hæmorrhage or peritonitis is considered an indication for its abandonment. The frequency of the repetition of the bath is governed by the thermometer alone, the object being to keep the temperature at an entirely safe point, 102° to 103° F. The temperature of the water at the time of the introduction of the patient into the bath should be about 90° F. It certainly should never be less than 65° F. If necessary, the water may be cooled while the patient is in the bath, but it should be remembered that a reduction to a safe point should be secured with water at the highest temperature which will accomplish the object.

The manner of practising the Brand treatment of typhoid fever is as follows: A bathtub filled about two-thirds full of water at a temperature ranging from 65° to 80° F. is placed by the side of the patient's bed, but from which it is separated by a screen, so that the invalid need not be annoyed by its constant sight or the preparations made from time to time for giving him the bath. On the patient's bed is placed a double blanket, covered by a linen sheet. Hot water bottles are kept handy for application to the feet after the bath. Prior to the immersion the patient is given a stimulant. He is then undressed, and if sufficiently strong he steps into the bath himself, otherwise he is lifted in gently by attendants. His first immersion may be accompanied by considerable nervousness and shuddering, but this readily disappears on reassurance. While immersed, every portion of the body with the exception of the abdomen is subjected to brisk frictions. This portion of the treatment is absolutely necessary, for it prevents chilliness, cyanosis and collapse. Attention to the pulse is important. Complaints of chilliness need not be regarded unless accompanied by chattering of the teeth, when the patient should be removed at once. The usual duration of the bath is fifteen minutes. When the patient is removed he is placed upon the sheet, which is carefully wrapped about him, and applied so that cutaneous surfaces are not permitted to touch. Then he is covered with the blanket. The hot bottles are applied to his feet. Von Ziemssen has modified the Brand treatment by what is known as the graduated bath. The water is at first prepared at a temperature of 86° to 90° F. With the patient in the bath it is gradually lowered in temperature by successive additions of water at a temperature of 40°, which must be thrown in so as not to come in contact with the body. The minimum temperature used is 72°. The duration of the bath is half an hour. Rubbing and chafing of the limbs and body are as necessary here as in the full Brand treatment.

As the temperature often continues to fall for a time after the patient is removed from the water, allowance should be made for the



expected reduction. The indications of the favorable influence of the abstraction of heat by means of cold water are a lessening of delirium, subsultus tendinum, tremor, and other nervous symptoms, an improvement in the circulatory system, more sleep, etc.

Fenwick has offered what appears to be a very useful substitute for the bath, in the device which he calls the ice-cradle. "This consists of an ordinary iron cradle sufficiently long to cover the patient in his entire length, and broad enough not to limit his movements and thus prove irksome. Under this the patient lies, covered by some light and opaque muslin. Attached to the cross-bars of the cradle are small zinc buckets in which ice can be placed. The outer surface of the buckets should be covered with lint to prevent any of the condensed moisture from falling on the patient. The cradle is covered with a counterpane, except at the two ends, which are left open to allow of a constant interchange of air. A hot bottle should be placed at the patient's feet, and before the cradle is used he should be well sponged with tepid water." Fenwick claims that by this means the temperature can be reduced as efficiently as by means of the bath. Ice is not always necessary. The continual surrounding of the patient's body by air is sometimes all-sufficient.

Medicinal antipyretics are allowable only in cases in which neither the homœopathic remedy nor hydrotherapy has been sufficient to control this symptom. The author has met with several such cases in which *acetanilid* especially has given excellent results. In suggesting this remedy it is with full consciousness of the present attitude of many, even of the allopathic school, towards temperature-depressing drugs. But history is simply repeating itself. It is the old story of excessive laudation, then repudiation. A valuable drug has in this instance been abused by prescribing it too freely in even the earlier stages of the disease, not to depress a dangerous temperature, but to prevent the temperature from attaining the danger point. The proper method seems to be the reservation of the medicine for an emergency which other means have failed to successfully combat. Indications of unfavorable action are sweating with some degree of collapsic symptoms, viz., enfeeblement of the heart indicated by feeble pulse and cyanotic surface. This can, however, hardly occur if the medicine is properly administered. Three grains can usually be safely given every three to six hours, if required that often, to control the high temperature. But it is preferable to give one grain each hour until the thermometer indicates a fall, then proceed cautiously no matter how slight the fall. It is never well to depress the temperature below what is considered a safe point, which is about 103° F. Used in this manner the medicine has in very small quantities proven sufficient to control the temperature at a time when a fatal result seemed inevitable. When quantities of three or four grains fail within a few hours to secure a reduction, larger doses have not proven satisfactory.

**CONSTIPATION.** There is too great a tendency, among homœopathic practitioners especially, to neglect the constipation of typhoid fever. Early in the disease care should be taken to have the bowels moved at least every second or third day. To accomplish this with the most ease to the patient and the least risk of interfering with other treatment or exciting the bowel lesion, inject into the rectum a few ounces of soapy water once daily. Large injections distend the bowel and often excite undue peristalsis. The small injection keeps the fecal mass soft and gently stimulates action at periods sufficiently often. The retention of feces for many days irritates the bowel, increasing the intestinal catarrh, raises the temperature, and tends to aggravate the oncoming intestinal symptoms.

**HEART-FAILURE.** The phrase "heart-failure" is at present a much abused one. It expresses a definite form of failure in certain acute forms of disease, but is often without meaning, as the failure of the heart referred to is only part of a general failure, and should not be singled out as a special feature. In order to be worthy of special attention and treatment, the failure of the heart should be determined to be more rapid than that of other systems. Whether due to mural degeneration or an impression upon the nervous apparatus of the organ, alcohol in some form is the remedy in most general use. The dose cannot be stated; it must be determined by observation in each case. Only enough to gradually improve the circulation is the golden rule. In some a quarter of an ounce every hour or two is sufficient. In others two or more ounces must be given within every three hours for a time. The early small dose should be increased until the desired result is attained, unless unfavorable symptoms arise, the diminished sensitiveness of fever patients to alcoholics being borne in mind.

Supposing the fever patient with a failing heart to be at perfect rest, and as perfectly fed as possible, and that the high temperature, severe cerebral symptoms or other possible causative conditions have received proper attention, alcohol in some form will now be given, and whether the stimulant is continued or not, a medicine must be selected. Those which experience has demonstrated as most valuable are *arsenic*, *agaricine*, *digitalis*, *hydrochloric acid*, *sulphate of sparteine* and *strychnine*. In urgent cases the hypodermic use of alcoholics, strychnine, atropia, etc., as recommended by some, may be of use, but personal experience has not demonstrated their value after the failure of other means.

**MANAGEMENT OF THE CONVALESCENT PERIOD.** Convalescence is established after the temperature has remained normal for two or three days. It is customary with most practitioners to gradually resume the use of solid food at this time. While this practice proves satisfactory in most instances it is equally damaging in others. It is better, even in the most favorable cases, to avoid meat and all articles of food containing fibre until the temperature has been normal for a week or ten days. The

presence of any degree of abdominal distension, tenderness, or of diarrhœa, is a positive contraindication to the use of solid food. It must be rare indeed that any unfavorable consequences follow upon the continuance of the fever dietary for one or two weeks succeeding the return of the temperature to the normal point, whereas serious consequences frequently result from the too early return to solid food. Stimulants in small quantities taken with the food are occasionally useful if the digestion is feeble. Exercise should be carefully regulated. Life in the open air is advised as soon the condition permits. Daily spongings with cold water and gentle massage favor the re-establishment of tissue nutrition. Should imprudence in exercise or diet lead to elevation of temperature the strictest regimen should be resumed at once, and continued until the patient is again in a favorable condition.

After the subsidence of the actual typhoidal symptoms there sometimes remains considerable prostration, with a slight evening rise, or morning subnormal fall in temperature. The ravenous desire for food which characterized the period of limited diet is replaced by want of appetite. In this condition of retarded convalescence a fresh trituration of *arsenicum iodatum* 2x, one or two grains every three hours, is of much value.

In the convalescent cases in whom prostration and weak heart have been pronounced, the amount of exercise should be graded. Perhaps a raising of the pillow a few inches per day until the sitting position is reached, will be necessary to avoid vertigo and faintness.

## TYPHUS FEVER.

**Nomenclature.**—This disease has received many names, Murchison having collated no less than ninety-eight. The most prominent of these are famine fever, ship fever, jail fever, spotted fever, putrid fever, etc. In the older German works it was called “exanthematic” typhus, to distinguish it from typhoid fever or “abdominal typhus.”

**Definition.**—Typhus fever is an infectious and contagious fever, endemic in certain countries possessing a crowded and poor population, and becoming epidemic during periods of general distress. It runs a typical course of about two weeks, and is characterized by a peculiar eruption, marked cerebral symptoms, and a termination by crisis.

**History.**—Murchison said truly that a complete history of typhus fever would involve a study of the history of Europe for the last three hundred and fifty years, during which period that division of the globe has been swept by numerous great epidemics of the disease. Up to the latter half of the present century these epidemics have always resulted in great loss of life; but with the beginning of that period they have gradually decreased in number and severity. Epidemics have several times invaded this country. In 1807 New England was visited by one. Philadelphia suffered in 1812, 1836, and the last time in 1866. During the late Rebellion the disease was not uncommon among returned prisoners of war. During the past decade typhus fever has several times obtained a foothold upon our Atlantic seaboard, but the cases thus developed have, fortunately, been few in number, and even these have been limited to the crowded regions of great cities.

**ETIOLOGY.** The exciting cause of the disease is exposure to a specific poison. We are probably justified in regarding this specific element as a microbe, notwithstanding careful investigations have as yet failed to discover a micro-organism which conforms to bacteriological laws relating to specific organisms. The predisposing causes are clearly established. They consist of every departure from correct dietetics and hygiene. Poor or scanty food, overcrowding, filth, want of a proper removal of the products of the human body, etc., are to be prominently mentioned in this connection. The disease is especially prevalent, therefore, in countries containing a large poor, crowded and filthy population, which exists in Russia, Hungary, Italy and other eastern countries, all of which play the part of endemic centres from which typhus spreads as a virulent epidemic. Outbreaks follow, as a rule, failures in the crops, or other influences calculated to still further deteriorate the condition of the people.



Intemperance, from its well-attested influence in producing systemic degeneration, acts as a powerful predisposing cause. Both sexes are affected by typhus fever with about an equal degree of frequency. Children are less liable to be attacked than are adults. The majority of cases occur between the ages of twenty and forty years. Although typhus has been generally regarded as a disease of cold weather, clinical investigations do not confirm this idea, as different epidemics have reached their height in the summer months.

The exciting cause of the disease is exposure to a specific poison. Typhus fever is highly contagious, and spreads rapidly wherever it breaks out. In garrisons it finds many victims. In private life it spreads from one member of the family to another; from the patient to the nurse; and from one household to others with which there is communication. The onward march of the disease can usually be easily traced, the poison being transported by individuals, clothing, and fomites generally. The specific poison from the patient is believed to be contained especially in the exhalations from the lungs and the skin, but the atmospheric diffusion of the same is not great, as those who keep a few feet from the patient are less apt to contract an attack than those who are in close proximity. Attendance on isolated patients is decidedly less dangerous than the care of numerous ones in a hospital ward. The danger seems to be in direct proportion to the degree of exposure. There is no contagious disease which attacks physicians with any greater frequency than this one, and yet they very often escape infection. Nurses, by reason of their constant presence in the sick-room, are far less fortunate. One attack of typhus fever affords an almost certain protection against others. In the expressive words of Moore it can be said that "the striking distance of typhus is not great." This is due to the rapid neutralization of the typhus poison by atmospheric air, a fact which has been positively demonstrated, the disease spreading little if patients are treated in tents, the ends of which are kept constantly open.

It has been said that the disease can originate in a community as the result of filth, overcrowding, etc., without having been carried thence by patients. Such a statement must be received with caution, being opposed to all positive knowledge concerning the cause of infectious disease. In all instances where such was apparently the case, it is probable that the germ of the disease had long existed about the spot, and only needed certain favorable agencies to render it active, or was imported in some unknown manner.

**Pathology and Morbid Anatomy.**—The general blood and tissue alterations present in typhus fever are almost identical with those found described as occurring in typhoid fever, exclusive of the specific lesion.

**BLOOD.** The principal alteration in the blood is a change in its fibrin, leading to lessened coagulability. The color of the blood is

darker than normal, and contains an excess of urea and ammonia, the latter probably resulting from the decomposition of the urea. The blood corpuscles are diminished in number with the progress of the disease, the outline in many is irregular, and large numbers present marked signs of degeneration. Much of the coloring matter of the corpuscles is separated from them, and it, with the altered blood, seems to filter through the degenerated vessel walls, leading to the accumulation of colored effusions and to staining of the tissues. The tissue changes are parenchymatous degeneration, fatty changes, and the so-called vitreous degeneration. These degenerative alterations are the causes of many of the clinical phenomena of typhus, the most prominent being such as are developed by means of the enfeebled muscular system, especially the heart. All of the internal organs are disturbed in the performance of their functions by reason of degeneration in the secreting cells, notably of the kidneys and liver.

**LUNGS.** Pulmonary changes are quite constant, the lung parenchyma being more frequently involved than the bronchial tubes. Hypostatic congestion and pleurisy, the various types of pneumonia and œdema, are all to be noted frequently.

**ABDOMEN.** The abdominal lesions are not at all characteristic. The glandular elements of the intestinal walls present evidences of congestion, but never of ulceration. If inflammatory changes and ulceration are found in the patches of Peyer, or solitary glands, it is positive evidence that the disease was typhoid fever and not typhus.

**GLANDULAR SYSTEM.** Certain cases are attended by an adenitis, which may be localized or quite general. This gland feature may characterize large numbers of cases during an epidemic, thus presenting a common and most interesting complication. The glands most frequently attacked are the cervical lymphatics, the parotid, sublingual and inguinal. The bronchial glands are almost constantly enlarged. Occasionally the bronchial glands have been found to contain pus. Any of the swollen glands may excite serious symptoms by reason of pressure upon adjacent tissues, as for instance, pressure of the axillary glands upon the brachial plexus, or the inguinal upon the femoral vein, or those about the œsophagus, interfering with deglutition. So great is the irritation and disturbance caused by these enlarged glands at times that they may determine a fatal issue.

**CRANIUM.** The most constant and characteristic pathological feature of typhus is congestion of the cerebro-spinal vessels. This condition stands in sharp contrast with the empty vessels and anæmic brain common to typhoid. The vessels of the brain, in some cases, are found engorged to such a degree that upon the removal of the skull they stand up in bold relief. In connection with the marked engorgement, or in certain epidemics in which engorgement is not so prominent, effusion is

found. The fluid is present especially upon the convexity of the brain. From five to ten ounces have frequently been collected. The effusion is clear, unless meningitis has complicated the case, when it appears as a more or less turbulent fluid, containing few or many fibrinous flocculi. There are no brain changes which cannot be ascribed to the pressure of the effused fluid. Sir William Jenner found hæmorrhage into the arachnoid in one-eighth of his fatal cases.

**Clinical Course.**—The stage of incubation is probably very variable. Murchison considered it to be about twelve days. In susceptible individuals the effect may be almost instantaneous. The onset of typhus fever is generally abrupt, the early symptoms being referable mainly to the nervous system, which is true of other infectious diseases. They consist of chills or chilliness, followed by fever, headache, vertigo, pain in the back and extremities, especially the thighs, anorexia, nausea, and constipation. Vomiting is unusual. The tongue is pale, furred and swollen. A high degree of prostration rapidly supervenes. If the patient still keeps about, notwithstanding these symptoms, the gait is staggering, the hands, the tongue, and the head tremble, and he is soon forced to take to his bed. The eyes are injected, the face flushed, and the pupils generally dilated. The nights are restless and disturbed by disagreeable dreams, and a mild delirium soon develops. By the fourth or fifth day a maculated, measly-looking eruption appears, and this becomes darker and more extensive with the progress of the disease. In six or seven days a well-marked petechial development occurs.

With the development of the symptoms of the second week, the pains are less complained of, due usually to the clouded intellect of the patient, for at this time delirium is usually well developed, being of a quiet, muttering, incoherent, or maniacal kind. This furious delirium may pass into coma, or a comatose state may gradually develop without previous active delirium. The interesting condition known as *coma vigil* is often present, *i. e.*, the patient is conscious while in apparent unconsciousness. Muscular tremor, subsultus tendinum, a dry brown tongue, sordes upon the teeth and lips, albuminuria, incontinence of urine and perhaps of the fæces, and all the symptoms of the typhoid state may now be developed. The temperature remains high, or is still more elevated; the pulse is rapid, often reaching 140 per minute; the respirations are increased; the hearing is dull; the intelligence is gone and the patient sinks towards the foot of the bed completely helpless. In this terrible condition he may pass into a complete coma and die, or if life can be sustained until about the fourteenth or fifteenth day, a critical change may occur, and this, at times, with astonishing rapidity. It is sometimes wonderful to see the helpless, almost hopeless sufferer regain his intelligence and rapidly lose the long train of serious symptoms.

The temperature, also the circulatory, respiratory, nervous and cutaneous symptoms call for more extended consideration.



**TEMPERATURE.** The early rise of the temperature is usually rapid, attaining from  $103^{\circ}$  to  $104^{\circ}$  F. within four or five days. There are exceptional cases in which the temperature is ten days or more in reaching the maximum. It occasionally happens that in some cases, even in those of great gravity, the thermometer does not record higher than  $103^{\circ}$  F. There is a daily morning remission of from one to two degrees. A marked remission often occurs somewhere from the eighth to the tenth days, followed by a gradual lowering of the morning and evening markings, until about the fourteenth day when a crisis occurs. This typical temperature range is subject to great variations due to so-called complicating conditions. Temperatures of  $110^{\circ}$  F. or higher have been reported as existing before death.

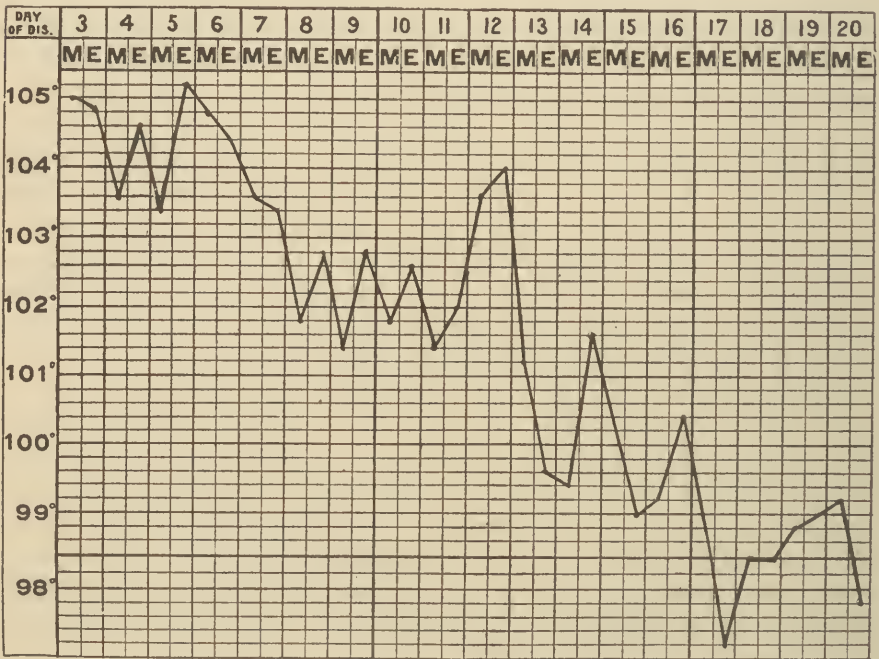


FIG. 5.—TEMPERATURE CHART OF CASE OF TYPHUS FEVER. (AFTER MOORE.)

**CIRCULATORY SYSTEM.** The pulse is seldom below 120, and weakens with each day of the progress of the disease. In serious and fatal cases the heart's impulse becomes almost or quite imperceptible, and the pulse irregular, intermittent and dicrotic, indicating low arterial tension. In exceptional cases the pulse may be reduced to 40 or 30. Murchison has cited cases in which the pulse ranged between 24 and 48 per minute. Such cases are always serious ones. In advanced stages embryocardia may be present.

**RESPIRATORY SYSTEM.** Bronchitis is almost constantly present from



the earliest stage, and cough is therefore a common symptom. In the second week hypostatic congestion or pneumonic consolidation may complicate the case. This is favored by position—lying on the back. These conditions are accompanied by increased and at times irregular respiration and some dyspnoea. Edema of the lungs may occur unannounced by cough or expectoration, but rather by rapid, difficult respirations, cyanosis, a cool, clammy skin, and ultimately coma.

**NERVOUS SYSTEM.** Headache is an early and very troublesome symptom, and lessens only when nervous symptoms of a higher grade overshadow it. In some cases it may be associated with rigidity of the muscles of the neck and retraction of the head. Active delirium seldom develops before the fifth or sixth day; prior to that time there are mental confusion, vertigo, and mild delirium especially upon awakening. The type of delirium depends much upon the character of the patient. Murchison and others have observed that the more intelligent the patient the earlier does the delirium appear, and the more serious is its character. As a rule the delirium is aggravated at night, and the patient is prostrated in the morning. Coma vigil, subsultus tendinum, tetanic rigidity of the muscles, grasping at flocks, picking at the bedclothes, loss of control of the sphincters, uræmic coma or convulsions, ringing in the ears, deafness, cutaneous anæsthesia or hyperæsthesia, are some of the more serious symptoms which may exist.

**ERUPTION.** The eruption appears upon the fourth or fifth day, and closely resembles that of measles. It may develop as early as the third or as late as the tenth day. It consists of dull red maculæ of differing size and contour, which fade insensibly into the color of the surrounding skin. A week may intervene between the appearance of the earliest and latest spots. Undeveloped spots give to the skin a mottled appearance—the so-called “subcuticular mottling.” This is a common but not an essential feature; and it is the appearance resulting from the presence of these two eruptive features to which Sir William Jenner applied the term “mulberry rash.” The eruption first appears about the wrists, upon the covered portions of the body, perhaps near the axilla, and later over the surface of the chest, back, arms and thighs. The eruption rarely appears upon the face, neck, or indeed upon uncovered portions of the body. This is probably due to exposure to the air. A moderate degree of development is also obscured by the cutaneous hyperæmia accompanying the fever. With the first appearance of the eruption the spots are slightly elevated, and may be effaced by pressure. They vary from one to several lines in diameter, but they become more and more persistent, under pressure, until finally they are unaffected by it. Each spot of this character now constitutes a petechia.

Purpuric spots and vibices are met in some malignant cases, or in typhus occurring in scorbutic individuals. These purpuric spots are

readily distinguishable from the petechiæ. If the eruption is quite dark or petechial it persists after death, not otherwise.

**Complications** are occasionally met with in the course of typhus fever. They are far more frequent in some epidemics than in others. Of these, those involving the respiratory system, the bronchi and the lungs are the most frequent. Pneumonia may be either catarrhal or croupous, and may end in gangrene.

The hæmorrhagic tendency of the disease is sometimes manifested by epistaxis, hæmatemesis, and, in exceptionally pronounced cases, by oozing from every orifice of the body, or even from the skin itself.

The parotid glands sometimes become inflamed, and this inflammation may proceed to suppuration. This is a serious complication.

The kidneys may become involved, nephritis sometimes appearing; but its presence is not necessarily indicated by the discovery of albuminuria, for that symptom is present in nearly all severe cases of the disease.

**Diagnosis.**—Typhus fever may be confounded with cerebro-spinal fever and typhoid fever. A knowledge of the existence of an epidemic of the disease is a great aid in enabling us to distinguish it from both of these affections, yet mistakes are not infrequent. During the last New York epidemic fully one-eighth of all the cases sent to Riverside Hospital as typhus were instances of mistaken diagnosis. Cases must necessarily occur in which the symptoms of one or the other of these affections will closely simulate those of the others. Typhus may present the occipital headache and rigidity of the neck of cerebro-spinal fever. In the former, however, headache is apt to be less severe, as are also the cervical rigidity and the hyperæsthesia of the special senses. Prostration is more marked in typhus.

The sudden onset distinguishes it from typhoid fever. The temperature curves in the two diseases are very different.

**Prognosis.**—This will vary greatly according to the genius of the epidemic. The mortality ranges from 10 to 50 per cent. Cases occurring in the young offer an especially favorable outlook. The habits of the patient modify the prognosis quite materially. Cases that recover run a course ranging, in the mild ones, from eight days, to twenty-one days in the severe ones.

**Treatment.**—As typhus is a highly infectious fever, the question of the best means of preventing its spread is the one which first obtrudes itself upon the physician. Among the poor, who can be but illy cared for at their homes, it is best to remove such patients to a hospital where proper care and treatment can be had, providing the patient is moved sufficiently early. A large percentage of the mortality resulting from typhus in hospitals is due to the late admission of the patients, necessitating a ride, often a jolting one, after the patient is unfit for removal;

also the probable neglect of proper care and treatment during the early stage of these cases.

In countries where typhus fever prevails, special institutions for its treatment have been established. When such special hospitals are not available, a large, airy, isolated ward should be selected, and the best means employed for securing rapid change of the air. There is considerable experience to show that much better results are obtained from the treatment of patients with typhus in tents, even in winter. The tents must be kept constantly open at their ends, allowing a current of air to pass constantly over the patients' beds day and night. Attendants should look after their own general health and avoid close contact with the patients. Physicians should not make physical examinations unless really necessary. All should have a full stomach when closely associated with the victims, as experience has seemed to show a lessened tendency to be affected by the contagion under such circumstances. It is wise to destroy the excretions, but the necessity for such action is not as imperative as in typhoid fever.

The general instructions given for the care, diet, etc., in the section devoted to the treatment of typhoid fever, are equally applicable to typhus. The absence of intestinal lesions in typhus fever, however, makes the excessive care in reference to diet there recommended, especially the exclusion of all solid food, unnecessary. Those who have seen most of this disease impress with emphasis the importance of a most nourishing diet. Milk, animal teas and extracts, long-cooked farinaceous gruels, eggs cautiously, blanc-mange, whey, wine whey, etc., are recommended. Drinks should be administered freely. Pure water is best, but for variety's sake, carbonized waters, cold weak tea or coffee, when not objectionable, are permissible. Water flavored with lemons, limes, currants, raspberries or tamarinds is admissible and often very acceptable.

There is a lack of clinical data upon which to build up a section upon the therapeutics of typhus. Our comparatively excellent results in enteric fever entitle us to considerable confidence in the treatment of typhus fever also. The explanation of this lack seems to be that the early confusion of several of the continued fevers prevented independent observations of typhus, while of late years the disease has been less frequently and extensively epidemic in countries where such observations were most likely to be made; also physicians have been evidently relying upon the groups of indications for remedies in enteric fever which have been accumulated with so much care. This is proper in so far as these remedies are symptomatically suitable to cases of typhus fever. But the difference in the pathology of these two fevers leads us to infer that the same remedies can hardly be of equal value in both. Intense hyperæmia of the brain is the most important pathological feature of typhus—while the brain of the enteric fever patient is generally anæmic;



both have delirium, even if this be principally toxæmic in both. The true typhus medicine must have this hyperæmia of the brain among its physiological effects. Theoretically we must recommend the narcotics strongly, as they represent the congested brain as well as the symptomatic peculiarities of the delirium and other cerebral symptoms. This is not the whole disease, however, important a feature as it may be, and we must in many cases in which the nervous symptoms are not the most conspicuous feature, select from another class of medicines. *Baptisia* is strongly recommended during the early stage by Hughes and other English physicians. It presents an excellent likeness of typhus in its general symptoms. Its special indications are well known. The tincture and lower dilutions seem most in favor. For the second week the mineral acids are more important, especially *phosphoric acid*. Jousset, Bähr, Wurmb and others, including many old-school authorities, recommend it, the latter believing it neutralizes ammonia, which exists in the blood in an unknown combination. *Phosphorus* is still more strongly urged for the cases of higher grade. *Rhus* and *arsenic* are recommended when the typhoid state is highly developed. It is especially during this week that medicines for the combating of special features and of complications must be selected: *belladonna* or *glonoin* for the intense headache and early hyperæmia of the brain; *hyoscyamus*, if these symptoms are unabated, and especially if delirium or coma supervenes; *opium* after *hyoscyamus* if the stupor increases and the general progress is unfavorable; *lachesis* may be of use in the same condition; *stramonium*, if delirium of a hilarious character, rather than stupor, has progressed unchecked by *hyoscyamus*; *hyoscine hydrobromate* or *agaricus*, especially the former, may prove useful for a high degree of delirium and sleeplessness which *hyoscyamus*, *stramonium*, etc., have failed to mitigate, giving one grain of the third decimal trituration hourly until relief is obtained.

*Antipyresis* is even more important than in typhoid fever, and is carried out according to the instructions there given. The baths should be begun in the early stage, and the temperature kept reduced to somewhere about 103° F. If impossible to employ baths, a rubber sheet may be spread under the patient and water used freely by sponging, douching, or from a small hose.

*Stimulants* are seldom called for in the young, but are invaluable at any age or period of the disease, and especially to those in middle or advanced life if there is great prostration, especially of the heart-muscle. Delirium indicates their employment, if not associated with a nephritis.

As a rule, alcohol is contraindicated if delirium exists in connection with severe headache, scanty albuminous urine, or any of the symptoms of uræmia. Some good observers, as Loomis in this country, do not strongly favor the use of alcohol in typhus, at least as a "plan of treatment," Loomis stating that it is rarely necessary if the patient can be freely



exposed to fresh air. An ounce of brandy or whiskey every three hours is the quantity and form most frequently employed. The influence of the alcohol must be carefully watched, and the quantity, or its further administration, determined by the effect. Alcohol should not be given simply because the disease is typhus fever, but prescribed, as every powerful drug should be prescribed, according to indications.

*Heart-failure* is to be treated as recommended for the same condition occurring in typhoid fever.

If the disease is well treated, *sequelæ* are not common.

## RELAPSING FEVER.

**Nomenclature.**—Febris recurrens; famine fever; recurrent typhus; secondary fever; bilious typhoid fever; hunger pest; spirillum fever.

**Definition.**—A specific infectious and contagious fever prevailing epidemically and caused by a spirochæte (Obermeier) and characterized by one to two or more febrile paroxysms of from five to seven days' duration, and remissions of a corresponding time.

**History.**—A relapsing form of fever was known to the ancients. The disease as we meet it in the present day was described in the early part of the eighteenth century, the first thorough account of it having been written by Rutty, describing the disease as it appeared in Dublin in 1739 and 1741. Epidemics of relapsing fever have frequently occurred in association with typhus. Before these forms of fever were differentiated, it was often stated that "the fever relapsed into typhus, or that typhus relapsed into a form without spots." The first authentic epidemic in America occurred in 1844, the infection being brought over from Europe in an emigrant ship. In 1869 an epidemic prevailed in Philadelphia. Aside from these occasions, but few cases have been observed on this side of the Atlantic.

Like typhus fever, relapsing fever is bred in filth and destitution. Its habitat is therefore Russia and Russian Poland, India, Ireland, Italy, and many eastern countries possessing a large poor population. Moore denies any great prevalence of the disease in Ireland, for though afforded exceptional opportunity for observing all febrile affections, he has never seen a case of it. The disease becomes epidemic after famine or any general causes calculated to further reduce the condition of the poor. Still, the well-to-do and the well-nourished are not entirely exempt.

**Etiology.**—Males are rather more frequently attacked than females. The susceptible period of life is that between fifteen and twenty-five years. The favoring conditions are such that the disease appears usually in cold weather. Overcrowding, lack of food or poor food, and filth are the predisposing trio, which seem essential to its epidemic prevalence. One attack does not confer immunity against further ones.

The specific element is the *spirillum Obermeieri*, a spirochæte discovered in the circulating blood of patients suffering from this malady, by Obermeier in 1873. The spirilla are found in the blood during the febrile paroxysm, but disappear gradually after its cessation, reappearing again with each relapse. They are motile in a marked degree. This micro-organism is obtained from the patient's blood, no matter what

the source whence it has been derived happens to be. It is absent, however, from all the secretions and excretions. The trysting-place of the spirilla during the stage of remission is largely a matter of conjecture. It is believed, however, that they then retire to the spleen. The disease is inoculable if blood taken during a paroxysm be used; also from pure cultivations of the spirillum Obermeieri. Vandyke Carter, of Bombay, has made careful experiments along this line. The spirochæte varies from 1-500 to 1-3000 inch in length, *i. e.*, from two to six times the breadth of a blood corpuscle.

During the remission, especially just preceding a relapse, small shining bodies appear in the blood. These are believed to be spores.

The disease is eminently contagious among those predisposed to it. Murchison has observed that epidemics of typhus and relapsing fevers are very apt to appear contemporaneously. In these mixed epidemics the relative proportion of cases of the two affections varies greatly at different times and places. The proportion of relapsing cases, however, is much greater at the commencement of the epidemic.

**Pathology and Morbid Anatomy.**—The blood presents, in less degree however, the same conditions as exist in infectious fevers of longer duration. During the paroxysms the liver and spleen are always much enlarged and hyperæmic, and at the autopsy are found to be friable and readily torn. The swelling rapidly diminishes during the intermission. The spleen is very often found at the post-mortem examination to be from four to six times its natural size. Its capsule is tense and often presents inflammatory areas of inflammation. The substance of the organ is intensely hyperæmic, soft, and even diffuent. Upon a cut surface are visible grayish or yellowish nodules, and the Malpighian corpuscles often necrotic or containing collections of pus. This suppurative process sometimes extends itself to large tracts of tissue, resulting in large abscesses. Rupture of the spleen has occurred in 5 or 6 per cent. of all cases (Peterson). Infarctions are frequent, and are believed by some observers to be due to obstruction of the splenic vessels by collections of spirilla. Others deny such occlusion. The liver presents evidences of granular and fatty degenerations. Peri-hepatitis occurs occasionally. The mucous membrane lining the gall-ducts is often in a state of catarrhal inflammation with resulting jaundice. The gall-bladder is often distended. In a "typhoid" variety of relapsing fever in which bilious symptoms are prominent, the liver is found post-mortem to be shrunken, the cells highly degenerated,—in fact, large numbers of cells having entirely disappeared, a condition closely simulating the appearance of acute yellow atrophy. Occasionally there is intense jaundice with staining of the tissues. Catarrhal inflammation of the alimentary tract is frequent. Swelling of the mesenteric glands has often been observed. Bronchitis and hypostatic congestion of

the lungs are present in nearly all cases, and the bronchial glands are enlarged. The kidneys are enlarged, their epithelia cloudy and swollen, and points of hæmorrhage are found in the cortex. According to Ponfick, the medulla of the bones contains foci of softened tissue as in the spleen, that may lead to the formation, in some instances, of abscesses, cysts, and caries of the bones. The changes in the nervous system are very slight. Œdema of the brain and hæmorrhage into the membranes have been observed. The muscles have a deep red color, with the exception of the heart muscle, which is generally pale, and numerous points of hæmorrhage may be present. The microscope shows extensive degeneration of the muscle fibres, more especially those of the heart. In general the muscles are dry and flaccid, or may be brittle and show evidence of cloudy swelling and fatty degeneration. Rigor mortis comes on rapidly and is very persistent. Ponfick, of Berlin, who has investigated relapsing fever with great care, claims that the constant enlargement of the spleen, changes in the blood, marrow of the bones, kidneys, liver and muscular system (especially of the heart) are, taken together, quite characteristic. This is in opposition to the teaching of most authors, that relapsing fever is without a characteristic pathological anatomy.

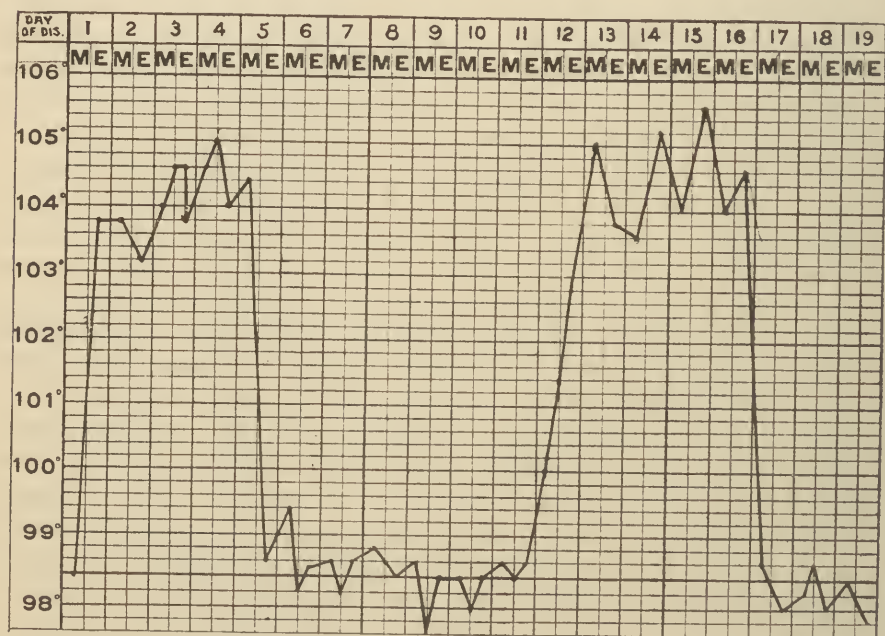


FIG. 6.—TEMPERATURE CHART OF CASE OF RELAPSING FEVER. (AFTER MURCHISON.)

**Clinical Course.**—The clinical features of relapsing fever are first a sudden and severe invasion; secondly, a stationary period or one of



increasing severity up to the termination of the paroxysm in a marked crisis which takes place some time between the fifth and the seventh days; and finally, an apyrexia of about the same duration as that of the febrile paroxysm.

The patient may be attacked apparently within a few hours after exposure in some cases. Usually, however, nearly a week intervenes between the reception of the infection and the appearance of symptoms. Prodromic symptoms are not marked, nor are they characteristic. Chills, fever and severe general pains are the trio ushering in the paroxysm. Nausea and vomiting are common. Convulsions or other severe nervous phenomena may be present in young subjects. Giddiness is usually a marked symptom. The temperature and pulse rise rapidly, the former often registering  $103.5^{\circ}$  to  $104.5^{\circ}$  F. within the first twenty-four hours, the latter from 100 to 120 or more per minute.

With the progress of the paroxysm, the temperature may rise even higher than above mentioned, registrations of  $105^{\circ}$  or  $106^{\circ}$  F. and even  $107^{\circ}$  being not uncommon. The face is flushed. The stupid appearance so often seen in typhus and typhoid fevers seldom appears, probably because of the short duration of the fever. Sleeplessness is almost always a very prominent symptom. The mind is seldom clouded, although a slight delirium may develop towards the close of the paroxysms. A maniacal delirium has been reported as coming on just before the crisis. The patient under such circumstances is exceedingly violent; his stools are involuntary, and in some cases there is a rapid elevation of the temperature. All these symptoms are speedily relieved by the crisis.

The crisis of relapsing fever is very characteristic. The fall is always a great and rapid one, even going several degrees below the normal. In extreme cases the entire fall may amount to from twelve to fourteen degrees, as in cases reported by Murchison, Pepper and others. Profuse sweating is common, while the menorrhagia, diarrhoea, or colapsic symptoms may sometimes be observed. A second and even a third relapse may occur, though but rarely. Each succeeding attack is shorter, as a rule, than its predecessor. Exceptionally, cases in which there is no relapse are observed.

During the epidemic of 1847 in Dublin, Stokes observed and studied a cardiac murmur of bellows character. In some cases there was a prolongation of the systolic sound. Especially were these cardiac symptoms observed during the relapses. This murmur Stokes regarded as of anæmic origin. Upon the day preceding the crisis, the thermometer may herald the approaching fall by showing a marked remission in the temperature. Frequently, however, the symptoms are greatly aggravated just before the crisis. After the crisis, the temperature remains subnormal for from one to three days. It then fluctuates about the

normal point until the advent of the second paroxysm, which usually takes place about the seventh day. Then there occurs a repetition of the symptoms and conditions observed in the original paroxysm. The temperature in the relapse is nearly always higher than during the first. This is, however, generally compensated for by its shorter duration. Its termination is by the same phenomena as those attending the initial paroxysm. After the third or fourth day, jaundice appears in about 10 per cent. of all cases. Percussion now demonstrates enlargement of the spleen and liver. Nephritis or pneumonia may appear as complications, and add their symptoms and signs to the disease.

**Sequelæ.**—Relapsing fever is a very exhausting disease. Convalescence is usually slow, especially if more than one relapse occurs. The more common sequelæ are pains in the limbs, involving particularly the regions of the knees and ankles, and the region of the long bones generally. The joints under such circumstances may be swollen. Other sequelæ are swelling of the lymphatics and of the parotid gland, boils, anasarca, and most characteristically, a peculiar form of inflammation of the eye first described by Dr. Mackenzie, of Glasgow, and called by him "post-febrile ophthalmitis." Estlander has demonstrated that this eye trouble begins in the choroid, extending thence to the vitreous body and sometimes to the iris. Recovery from it is slow, it sometimes requiring two months before sight is restored. Permanent blindness may result from it. Pneumonia is a frequent and dangerous complication, though by no means a necessarily fatal one. Certain circulatory changes afford a subject of melancholy interest. Owing to the weakness of the heart muscle, cardiac thrombosis becomes a cause of sudden death. Embolism has been observed with considerable frequency. It may occur in any portion of the body or internal organs and may be the means of producing localized gangrene. Hæmorrhagic complications, as hæmatemesis, though not common, are far from rare and are always very dangerous symptoms, the majority proving fatal. Diarrhœa sometimes complicates the disease, though not with the frequency observed in typhoid fever. Dysentery is especially liable to occur in the East Indian epidemics.

**Diagnosis.**—After the presence of an epidemic is determined, the diagnosis is easy. The peculiar temperature curve and the symptoms associated with its rise and fall are sufficiently characteristic. In questionable cases the blood should be examined for the spirochæte.

**Prognosis.**—When we take into consideration the intense character of the pyrexia, the frequently serious nature of the anatomical change and general symptoms, the impaired vitality of those attacked by reason of want and bad surroundings, we are surprised at the small number of persons who succumb to relapsing fever. A review of the more recent collections of statistical reports indeed does not leave the disease in the

category of "mild fevers." In reporting the mortality in this disease, too much stress has been laid on the few deaths in uncomplicated cases, excluding those presenting jaundice, pneumonia, nephritis, etc. Thus Murchison, in his classical treatise on "Fevers," reports 2,115 cases admitted to the London Fever Hospital in twenty-three years, with a mortality of 1.84 per cent. He also analyzed nearly 17,000 cases occurring in Scotland and found a mortality, in that country, of between 4 and 5 per cent. In 103 cases admitted to Bellevue Hospital, New York (epidemic of 1869-70), two only proved fatal. In some epidemics, however, there is a much higher mortality, *e. g.*, in the Philadelphia epidemic reported by Pepper the mortality was 14.4 per cent. among 1,174 cases collected. Carter, in an epidemic observed in Bombay, reported a mortality of 18.02 per cent. among a collection of 616 cases (111 deaths). These statistics are sufficient to show that while in general the number of deaths is small, in certain epidemics the mortality causes relapsing fever to rank with typhus and typhoid fevers. Among observers of our own school, Hahnemann treated 183 cases in Leipsic in 1814, without a single death. Dr. Kidd in the Irish epidemic of 1847 treated 111 cases of fever, of which number he considered 24 to be typhus and 87 relapsing fever. Two died, both supposed to be typhus. If his judgment was correct, his mortality in relapsing fever was nothing. Dr. Dyce Brown, of Aberdeen, in 1871 treated 50 cases without a death.

Death occurs at any period of the disease, *i. e.*, during the primary paroxysm, apyrexia or the relapse. It may be due to the intense pyrexia and serious attending symptoms: prostration, collapse due immediately as a rule to degenerate heart muscle, or to certain of the so-called complications, as pneumonia, hæmorrhage, jaundice, liver lesions, thrombosis, hepatic or splenic abscess, uræmia, etc. Unfavorable symptoms in relapsing fever are jaundice, especially when conjoined with cerebral symptoms, purpuric spots, copious hæmorrhages, suppression of urine and the typhoid state.

**Treatment.**—The general treatment should be conducted upon the principles followed in the management of the infectious fevers generally, *i. e.*, isolation, absolute rest in bed, liquid diet, and the best hygienic surroundings. That the disease may be carried by fomites must not be forgotten. The paroxysms can be modified somewhat by treatment, notwithstanding Osler's most positive statement to the contrary. That authority makes the bold assertion that the paroxysms themselves cannot be modified, and their recurrence cannot be prevented by any treatment whatsoever. On the subsidence of the fever, the patient requires a more liberal dietary than is wise in other exhausting fevers as typhus and typhoid. During the collapse following crisis, alcoholic stimulants may often be administered with signal advantage.

Such uniformly excellent results have been secured by several

observers, each using in the main a single remedy (Hahnemann recommended *bryonia* and *rhus*; Kidd, *bryonia*, Dyce Brown, *baptisia*), that we may well make considerable claims for the action of medicines in this peculiar disease. These reports above quoted show most conclusively that the homœopathic treatment is more successful than that of the dominant school. In an epidemic observed in Albany, Dr. H. M. Paine found that *chamomilla* had a modifying effect on the paroxysms.

During the stage of invasion, *aconite*, *bryonia* and *veratrum viride* have been recommended; with the full development of the fever, *bryonia*, *rhus tox.*, *baptisia*, *cimicifuga*, and *eupatorium perfoliatum*.

Dr. Dyce Brown recommends *arsenic* for the watery diarrhœa and vomiting in the early stages, and says it signally meets these symptoms. The elder Guernsey used *arum triphyllum* with good effect, guided by symptomatic indications. For the post-febrile ophthalmitis, a most liberal dietary is advisable.



## DENGUE.

**Nomenclature.**—This affection has received many names, most of them being fanciful, and based upon certain features of the disease, or upon theories as to its causation, pathology or region of prevalence, etc. The negroes of the West Indies called it “dandy fever” on account of the rigid carriage of those afflicted with it. Dengue is probably a Spanish corruption of “dandy.”

**Definition.**—A specific infectious disease presenting fever, violent articular and muscular pains, and, generally a cutaneous rash. Dengue has an average duration of one week, and presents three periods: (1) An initial fever, the paroxysms lasting three or four days and followed by (2) a remission, which is often absent, and (3) by a terminal febrile paroxysm. Relapses occur even after several weeks have intervened, but are usually milder than the primary seizure.

**History.**—According to Zuelzer, our earliest knowledge of this disease dates from the latter part of the eighteenth century (1779), when it prevailed in Java. Dr. Rush described the epidemic as it appeared in Philadelphia in 1780, and Dr. S. H. Dickson, of Charlestown, S. C., published an account in 1839. Subsequently to that time his contributions to the subject have been looked upon as authoritative. The disease is generally epidemic and is confined to tropical and semi-tropical countries, its usual limits being latitude  $32^{\circ}$  N. and  $22^{\circ}$  S. Repeated epidemics have occurred in our Southern States during this century. The susceptibility of a community in which the disease is epidemic is truly remarkable, so few persons escaping. It is believed that the domesticated animals may be attacked by dengue, for often during the course of an epidemic they suffer from what is apparently an analogous disease.

The most recent epidemic, that of 1880, was probably the most extensive this country has witnessed, few of the large cities of the South escaping a visitation.

**Etiology.**—Little is known of the cause of this peculiar affection. Since the prevalence of the germ theory, it has been ascribed to a micro-organism. It is considered infectious, and by some contagious. There is abundant evidence of the portability of the poison. It prevails during warm weather, being dissipated by the frosts. The population living by the sea and upon low lands are most affected. All ages and both sexes are alike attacked. Permanent immunity is probably conferred by one attack, though this statement has been denied by competent authorities, who claim that the contrary is the case, and that one attack predisposes to another.

**Pathology and Morbid Anatomy.**—The infrequency of a fatal issue has made the pathological study of this disease almost impossible. The tissues about the affected joints are found to be infiltrated with a serous fluid. In some cases, too, effusion occurs within the sheaths of the tendons. McLaughlin, of Texas, has made strong claims concerning certain micrococci, which he has found in the blood of dengue patients, and has published elaborate arguments supporting the theory that the same are the specific cause of the disease, but his observations have failed of confirmation by bacteriological authorities.

**Symptoms.**—There is a period of incubation covering a few days. Prodromata are not frequent, but if present they are pretty much the same as those of other infectious diseases. The onset is therefore sudden, and marked by chilliness, headache, photophobia, anorexia and intense pain in the joints or muscles. Sometimes this pain constitutes the initial symptom. With the development of the pain the temperature and pulse rise, the former to from 101° to 107° F., according to the intensity of the attack. There are the usual attending conditions of fever. The pain increases during the first three days, usually disappearing by the fifth day. Then the temperature falls, and a stage of remission is established during which the patient complains of stiffness, soreness and weakness; this crisis being often attended by profuse sweating or diarrhœa. The eruptive manifestations may take place at any stage of the attack, but generally somewhere from the third to the fifth day. There is nothing distinctive about them. The form of the eruption may be scarlatinous, measles-like, papular, urticarial, herpetic, etc. There is more or less of irritation of the skin attending the eruption. There often follows dry desquamation, accompanied by considerable itching. In slight cases, the eruption may be absent. Both the large and small joints may be affected, and manifest varying degrees of swelling, redness and pain. Hæmorrhages from the mucous membranes may occur, and black vomit has been noticed. The mucous membranes lining the mouth, nose and throat may be inflamed, and the lymphatics swollen. The glandular enlargement often continues long after convalescence is completed. Children may have convulsions at the onset, and they are frequently delirious. This occurs but seldom with adults. In some cases special organs or tissues bear the brunt of the disease. Thus we have the gastro-intestinal type, in which vomiting and purging are the prominent manifestations. In other cases brain symptoms predominate. These latter must, however, be regarded as the result of the high fever rather than of actual cerebral inflammation.

The degree of prostration resulting from an attack of dengue often seems out of all proportion to the intensity of the disease. Convalescents are often unable to endure mental or physical exertion for weeks. The mild cases recover rapidly, the fever paroxysm being light and

short, and the disease really giving up its grasp with the termination of the initial febrile paroxysm. The violent pain is always a distinctive feature of this affection. The skin eruption may be followed by desquamation. Deaths are almost unknown, and we are, therefore, ignorant of the morbid changes existing.

**Diagnosis.**—Occurring as an epidemic, and attacking, as has been stated, all classes of people, there can be but little difficulty in making a correct diagnosis. Sporadic cases might be mistaken for inflammatory rheumatism, and, if yellow fever were prevailing, confusion might also result from this source. The intense pain attacking, notably, the joints, but diffusing itself over the back and limbs, as in infectious diseases, is the most important diagnostic feature.

Influenza bears quite a number of resemblances to dengue, but also presents a variety of important distinctions. In the former affection, herpes is the only eruption; the joints are not affected, and when the fever disappears, it does so permanently, unless complications ensue. Influenza may, moreover, occur in any clime, and its victims are remarkably prone to a variety of sequelæ.

Certain forms of dengue, with eruption as the main feature, may present diagnostic difficulty. DeBrun, who has given this subject considerable attention, divides the eruptive forms of dengue into three classes, as follows: (1) *The febrile eruptive form*, which presents important differences from the eruptive fevers in that the eruption is not always present; and when present, does not manifest itself at any fixed stage of the malady, and its appearance does not produce any modification of existing symptoms. (2) Another variety is the *apyretic eruptive form*, characterized by an eruption appearing over the whole, or a portion, of the body in direct proportion to the severity of the other symptoms, but without any rise in temperature. (3) The *exclusively eruptive form*, in which the rash is the only symptom.

The characteristics of the dengue eruption serving to distinguish it from other ailments are: It appears just as frequently on the chest or limbs as on the face; it is never associated with nasal or bronchial catarrh; it rarely appears with the onset of the fever, and is nearly always followed by desquamation and itching.

**Prognosis.**—This is very favorable indeed, death being exceedingly rare. Some few cases, occurring in infants, die from convulsions; and in adults, complications occasionally claim a victim.

**Treatment.**—Rest in bed is necessary. The diet should be of a liquid character. The patient may be permitted cooling drinks. The remedy indicated will generally be found to be *aconite*, *gelsemium*, *arsenicum*, *rhus*, or *eupatorium*.

Therapeutic suggestions must be based largely upon the authority of our Southern confreres, who alone have had extensive experience in

this disease. Falligant (Arndt's "System of Medicine," Vol. III, p. 374) speaks as follows: "As internal remedies, *aconite* and *bryonia alba* are specially called for in the first stage, with *ippecacuanha* to relieve vomiting, and *arsenicum album* for diarrhœa. If fever proves obstinate and the eruption is out on the skin, *bryonia* and *rhus tox.* are most useful. The disturbances of the stomach, such as flatulence, nausea, etc., call for *colocynth* and *nux vomica*. Sulphuric acid, *arsenicum*, *secale cornutum*, and *china*, sometimes followed by *ferrum carb.*, are all needed in different hæmorrhagic conditions. Where there is a hæmorrhagic diathesis it will be found advantageous to give internally a watery solution of tincture of *ferrum chlor. fort.* every two or three hours. Renal hæmorrhage requires *cantharides*, *belladonna*, *arsenicum*."



## MILIARY FEVER.

**Synonym.**—Sweating-sickness.

**Definition.**—An acute apparently infectious disease occurring in epidemics, and characterized by fever, profuse sweating, epigastric distress, and a papulo-vesicular eruption.

**History.**—The first historical outbreak of this disease occurred in 1486, in which year the army of Henry VII returning from the battle of Bosworth Field was attacked by the scourge. The disease then rapidly became epidemic throughout England. Since that time, numerous other epidemics have occurred, but these have gradually become less and less widespread, and the territory liable to them, more and more limited. At the present day, only portions of Italy and northeastern France seem to possess any liability. Even there the epidemics prevail in small villages and communities only.

**Etiology.**—Absolutely nothing reliable is known concerning the causes and nature of this peculiar disease. It is believed to be non-contagious, and yet it spreads rapidly in a community. Most epidemics have occurred during the spring and summer months. Overcrowding, bad hygiene, and the usual surroundings of poverty do not predispose to it. It usually appears in districts characterized by low damp ground, and yet it has invaded high-lying and dry places. People in middle life furnish the greatest number of victims.

**Symptomatology.**—The usual prodromal symptoms of the acute infectious fevers, namely, malaise, prostration and headache, usher in this disease. Then the patient is suddenly seized with fever, profuse sweating, and most intense distress in the epigastrium. It is the latter symptom that occasions the most pronounced source of suffering in this disease and it is associated with more or less abdominal tenderness. The temperature is rarely very high; but the pulse is quite rapid. About the third or fourth day of the disease the eruption appears. This consists at first of red spots of irregular outline and varying size, being generally from one- to three-sixteenths of an inch in diameter. Within a few hours of their appearance, a vesicle forms in their centre. The contents of these vesicles soon become opaque. In the course of two or three days they dry and form crusts which fall off leaving a normal surface. The individual lesions may be either discrete or confluent. The eruption appears generally first upon the neck and chest.

When the attack is a severe one, as quite often happens, severe symptoms of asthenia appear. These are weakness with insomnia, vertigo, headache, nausea, vomiting, and convulsions.

**Diagnosis.**—From rheumatism miliary fever is to be distinguished by the absence of swellings of the joints; from measles by the vesicular character of the eruption and the absence of catarrhal symptoms; from malaria by the absence of periodicity in the febrile manifestations. In any case the knowledge of an epidemic prevalence of the disease is a valuable aid to diagnosis.

**Prognosis.**—This varies with the epidemic. The influences which tend to gravity of the disease are unknown. In some epidemics the fatality is practically nothing. In others it is remarkably high, as great indeed as 80 per cent.

**Treatment.**—This must be conducted both hygienically and medicinally on symptomatic indications and general principles. Very little reliable information concerning the therapeutics of miliary fever is at our disposal. This remark applies to both schools of medicine.

## INFLUENZA.

**Nomenclature.**—The term influenza is of Italian origin (signifying influence) and is the name most generally accepted. Its use was founded upon a fanciful notion as to the supposed influence exerted by the atmospheric air or the stars in the development and spread of the disease. The recent epidemics have been known as la grippe (the laity designating it “grip”). The origin of this term is the French verb *gripper* (to seize). The origin of epidemics in various countries at different periods has led to the appellations Russian fever, Spanish fever, etc.

**Definition.**—Influenza is an acute infectious fever, in slight degree contagious, which is sporadic in most communities, becoming occasionally epidemic, and at intervals of years spreading as a great pandemic over large portions of the habitable globe. The most prominent symptoms are fever, catarrhal inflammation, most frequently of the respiratory tract, less often of the gastro-intestinal mucous membrane, prominent features due to the influence of the poison upon the nervous system, and a wide array of complications and troublesome sequelæ.

**History.**—Epidemics of this disease have swept the world for ages. The first accurate descriptions of it date back to the sixteenth century. The first recorded American epidemic was in 1655, since which time we have had quite numerous visitations from it. The last great epidemic which is fresh in the minds of all, and which was so general as to be pandemic, started in Bokhara in May, 1889, reaching Russia in October of the same year. It spread rapidly through Europe, invading England and America in December, reaching its height in the latter country in January and early in February, when it rapidly declined. Fresh outbreaks occurred in the winter of 1890–91, and sporadic cases were observed in 1891–92, also in 1893–94. The 1889–90 epidemic was remarkable for its universality. In many large cities so many were ill with influenza that business was very seriously crippled. The general death-rate was greatly increased, in many places doubled, although the number dying of influenza *per se* was very small indeed.

**Etiology.**—There is much concerning the etiology of influenza that is not capable of explanation in the present state of our knowledge. We know that it may occur either epidemically or sporadically, and we have every possible reason for believing that it depends for its existence upon a micro-organism, especially from the analogy existing between influenza and the acute infectious diseases. Such an organism Pfeiffer, of Berlin, discovered in 1892. His observations appear to have been sufficiently cor-

roborated by other bacteriologists of note to establish their correctness. This organism is a bacillus with rounded extremities, nearly as thick as the bacillus of mouse septicæmia, but only half its length. It does not appear motile, nor could a capsule be demonstrated. Cultures were obtainable upon agar direct from the sputum, and growing at temperatures of 78.8° to 80.6° F. Further developments were impossible without the presence of blood, especially of the hæmoglobin, indefinite propagation being possible under these conditions. The colonies were small with a peculiar vitreous transparency, and without resulting liquefaction. The organisms are inflexibly aerobic. They are very sensitive to drying, but retain their power of infection for at least two weeks if kept moist in sputum. Spores have not been demonstrated. Pfeiffer believes they do not exist. Contagion is probably transmitted by means of the moist secretions of the respiratory mucous membrane. In the early stage, the organism is found free in large numbers, later, in larger and larger numbers within the pus cells. They have not been found in the blood. The organism stains well in Löffler's methylene-blue fluid, the composition of which will be found in the chapter on anthrax and also in diluted Ziehl-Neelsen fluid, the composition of which is as follows: 10 cc. of concentrated alcoholic solution of fuchsine are added to 90 cc. of a 5 per cent. solution of carbolic acid. If the staining fluid be warm, the specimen can be prepared in a few minutes. Cultivated on glycerin-agar and allowed to solidify obliquely, they develop an appearance as of little drops of water, which require the assistance of a magnifying glass for their detection.

For the detection of the bacilli in the blood, Canon gives the following directions: The blood is put on the cover glass and dried in the usual way for bacteriological examinations. The cover glass is then treated with absolute alcohol for at least five minutes, after which it is allowed to remain for from three to six hours in Chenzynsky's eosin-methylene-blue fluid, which is as follows: Concentrated watery solution of methylene-blue, 60 parts; one-half per cent. of eosin in 75 per cent. alcohol, 20 parts; distilled water, 40 parts. To this add 12 drops of a 20 per cent. solution of caustic potash.

A "pseudo-influenza" bacillus has also been discovered, present in the sputum of individuals when influenza is not prevalent. It is somewhat larger in all dimensions than the influenza bacillus, and forms into chains. Its behavior with staining fluids and under cultivation suggests that these bacilli—*i. e.*, the pseudo-bacillus and the bacillus of influenza—may belong to one group. The true bacillus is present only in influenza, and its numbers bear a relationship to the intensity of the attack, with the subsidence of which it disappears. Inoculation of apes and rabbits with the true organism has resulted in the development of quite typical attacks. A stumbling-block to a belief in the dependence of influenza



upon a specific organism is the rapid, perhaps simultaneous development of the disease at many widely separated foci; these facts of development not according with our knowledge respecting the spread of pathogenic organisms, it is proposed to overcome this objection by assuming that the bacillus of influenza is a widely distributed organism, under ordinary conditions giving rise to occasional sporadic cases of the disease, but that, owing to general conditions not understood, it attains a virulent quality of infection to which most persons succumb. Under these circumstances the influenza bacillus is second to no organism in its ability to produce its specific effects.

Whether or not influenza is contagious to any great extent is as yet unknown. The disease has apparently been carried by the corpse of a patient dead with influenza, and it is believed that it can be carried by fomites.

Most authorities teach that it occurs independently of seasonal or climatic relations, and that elevation or character of soil exert no influence on its appearance or spread. There is a general impression, however, that during the late epidemic, cases were more frequent and of more severe character during the damp, murky weather of that memorable winter, and that the general health-standard of the people was better, the amount of illness less, and susceptibility diminished when dry, cold, seasonable weather prevailed.

All persons were alike susceptible to the infection; children, however, less than adults. The aged and those depressed by fatigue, anxiety, and general bodily ill-health, were not only more liable to contract the disease, but were very severely affected when attacked.

**Morbid Anatomy.**—The most common feature of influenza is a catarrhal condition of the mucous membrane of the respiratory and gastro-intestinal tracts. The capillary bronchial tubes may be involved, and both the lobular and lobar forms of pneumonia are common complications. Fibrinous and purulent pleurisy and pericarditis have been frequently recorded, also meningitis, or cerebro-spinal meningitis; neuritis is common. The pathological appearances in the various complications differ little or not at all from the same diseases when appearing as primary affections. Although without a specific lesion the pathological ramifications of influenza are exceedingly widespread. One has only to scan the journal literature of the past few years to feel that the influenza poison has been credited with being the exciting cause of a vast, really unmentionable number of lesions, developed as complications and sequelæ.

**Clinical Course.**—Influenza is usually ushered in suddenly with high fever, associated with catarrhal symptoms, general aching, severe headache, general pain and profound nervous depression. Symptoms differ in individual cases according as the catarrhal or the nervous

symptoms predominate, and also according to the localization of the catarrhal process. This leads to the classification of the cases into two varieties, the catarrhal and the nervous: the catarrhal being further subdivided according as the mucous membrane of the respiratory or of the gastro-intestinal tract is involved.

(1) CATARRHAL VARIETY. This is ushered in with high fever, the temperature reaching perhaps 103° or 104° F., is attended by coryza, conjunctival injection, and severe paroxysmal painful cough, with scanty but tenacious sputum. Attending these symptoms are severe headache and myalgic pains, and usually a *profound depression altogether out of proportion to the intensity of the disease*. Sleeplessness is often both persistent and distressing. Delirium is rare. Physical examination of the chest reveals quite a number of irregularly distributed râles. If there are no complications and the patient has been properly cared for the fever disappears by crisis in three or four days, the patient remaining weak. *There persists a strong tendency to renewal of the catarrhal symptoms from very slight exposure or unusual exertion.*

In other cases the mucous membrane of the *gastro-intestinal tract* bears the brunt of the disease. The general symptoms above described are present, but in addition there are nausea, tenderness in the epigastric region, abdominal tenderness and diarrhœa. The onset of this type is abrupt.

(2) THE NERVOUS VARIETY. This type rarely exists free from the catarrhal element. The symptoms consist of sudden accession of fever, agonizing headache, hyperæsthesia of the special senses, delirium, pains in the back and legs, and irregular and slow pulse and respiration. Prostration is great and persists during convalescence.

**Complications, Sequelæ, etc.**—The remarkable tendency to catarrhal inflammations on the slightest exposure explains most of the complications of influenza. The most prominent and important, as well as fatal of these, are bronchitis and broncho-pneumonia. The bronchitis may involve either the larger or smaller tubes, and by extension may lead to pneumonia. Croupous pneumonia may also occur. In some cases the pneumonic condition may come on without apparent exciting cause, showing itself by sudden accession of fever, followed by the physical signs of pneumonia. In most cases carelessness is at the foundation of the trouble. During the late epidemic in Philadelphia pneumonia occurred in about 4 per cent. of the cases, and 11.65 per cent. of those proved fatal. The old and the feeble are especially prone to this complication. Homes for the aged, especially, have presented a high mortality from this cause. I met with abscess and gangrene several times during the past epidemic.

Other lung troubles are not by any means unknown as sequelæ of influenza; thus we may have empyema, plastic or serous pleurisy, etc.

Patients suffering previously from chronic bronchitis or pulmonary phthisis are always very unfavorably affected by influenza, which either intensifies the primary disease or hastens its downward progress. The mortality of influenza among phthisical patients is high.

Cardiac complications are frequent. Ordinarily these consist of marked weakness, a condition that has by some been attributed to unwise dosage with antipyrin and other antipyretic drugs. Purulent pericarditis has been referred to.

Nervous symptoms are very frequently observed as sequelæ. Prominent among these are those groups of symptoms constituting that widespread disorder, neurasthenia or nervous exhaustion. More tangible troubles are persistent headaches, insomnia and neuralgias of various kinds. Mental disorders are liable to occur, and may take the form of acute delirium, melancholia or simple failure of the mental faculties. The acute manias generally manifested themselves very shortly after convalescence.

In some of the cases of the nervous type the symptoms partake strongly of those of cerebro-spinal meningitis of the epidemic form. Indeed it may well be questioned if these cases do not have as an anatomical substratum, inflammation of the cerebro-spinal meninges. These diseases have been long known to prevail in close connection with each other. Many cases are so closely allied by symptoms that only the development of bacteriological methods of differentiation can be hoped for as a means to a certain diagnosis.

Among the late sequelæ often noted are various nerve pains and paralyses, which are undoubtedly due to a perineuritis.

**Diagnosis.**—During the prevalence of an epidemic the recognition of influenza is a very simple matter. At other times one is obliged to rely upon the general symptoms, the pains in the back and legs, the headache and the profound prostration. In the gastro-intestinal form the symptoms may simulate those of typhoid fever, but are differentiated from that condition by their sudden accession, and the marked catarrhal manifestations. Then, too, typhoid fever is associated with enlargement of the spleen and the characteristic eruption.

The general mortality of a community is greatly increased during the prevalence of an epidemic of influenza, this disease selecting the old, the feeble, and those who are subjects of chronic disease for its victims. While an uncomplicated influenza is readily distinguished, it is not so with cases in which complications develop early, particularly pneumonia. The type of pneumonia associated with influenza is identified by the more gradual onset, the frequent absence of chill, the precedence of catarrhal symptoms, associated with the general sense of prostration, etc., common to influenza. The signs of lobar consolidation do not increase as rapidly nor become as pronounced, as a rule, as in pneumonic fever.



The nervous form may present symptoms which resemble in a striking manner cerebro-spinal fever, and a diagnosis is impossible if the catarrhal symptoms have not preceded those related to the cerebro-spinal system. Bacteriological science must come to our help here.

**Prognosis.**—The prognosis of uncomplicated influenza is very favorable, but few cases dying. But if carelessness in the management of the patient is permitted the mildest case may result disastrously, not only as to undermining of the general health, but as to life. The effects of influenza are especially dangerous in the aged and infirm, and in the subjects of chronic respiratory complaints, especially of phthisis. In the latter it often results fatally or accelerates the progress of the disease.

**Treatment.**—No matter how mild the case or how slight the elevation of the temperature, the place for the influenza patient is in bed. There he should remain until well if he wishes to avoid speculation upon his health. Until his wonted physical condition is restored the greatest attention should be directed to the avoidance of exposure and overexertion. In general, the fever need receive no special attention other than that resulting from the general medication according to the totality of the symptoms. The tar products may be blamed for many bad results.

In the early stage of influenza *gelsemium* is generally the most efficient remedy. A careful study of this drug will convince one of its applicability to the initial symptoms. Ten drops of the tincture in four ounces of water, and teaspoonful doses hourly, is the best method of administration. *Aconite* may be more serviceable if the onset is sthenic in character. From either of these medicines one must, in active cases, soon turn to remedies better suited to the fully developed disease. Prominent among these is *bryonia alba*, which is an efficient medicine when the bronchial mucous membrane is attacked and the general pains are tormenting. It is undoubtedly one of the most important of the general medicines for influenza. If the general pains are very distressing, in spite of the use of the bryonia, *rhus toxicodendron* may give better results if the pain causes great bodily restlessness. *Eupatorium perfoliatum* is preferable if the upper respiratory tract is particularly attacked and accompanied by great pain and soreness of the whole body. The pain excites constant motion. This medicine is much more efficient in five-drop doses of the tincture repeated every one to three hours. If not relieved by remedies of this class, *antipyrin* in frequently repeated doses of the one-tenth dilution often relieves magically, especially if the pains are of a shooting character. *Phenacetin* in doses of three grains may be considered for intense pains, but larger doses had better be avoided. *Morphine* is preferable, as continued large doses of the tar products tend to the development of cardiac debility.

The bronchial and pneumonic features demand the same treatment



as when occurring as independent affections. For the bronchitis with heavy muco-purulent expectoration the *iodide of antimony* is pre-eminent. If the expectoration is copious, and unchecked by the iodide of antimony, the *iodide of tin* has succeeded. *Bichromate of potash* is occasionally indicated by the character of the expectoration and acts well. Each of these remedies is advised in the second decimal preparation.

For the treatment of the pneumonias of influenza, it is hardly necessary to do more than refer to the sections upon pneumonic fever and catarrhal pneumonia. *Phosphorus*, *tartar emetic*, and the *arsenite of antimony* are especially valuable.

For influenza in old or feeble persons, with great prostration, *arsenic* is a remedy highly prized. If bronchitis or any inflammatory complication is a marked feature of the case arsenic is contraindicated. It seems rather suited to cases in which the symptoms of general poisoning predominate rather than those of local changes.

*Salicylate of sodium* has been much recommended for "grip," by some, in large doses, by others in two or three grain doses frequently repeated. It possesses considerable influence over the pains and may be suitable when the disease attacks rheumatic individuals.

The perineuritis must be treated by rest, galvanism, massage, and the *chloride of gold* in doses of the  $\frac{1}{100}$ th grain, four times daily; counter-irritation may assist. In the treatment of most of the sequelæ, when admissible, change of air will be found of benefit.

Gastro-intestinal disturbance is often controlled by *ipecac* in small doses. *Arsenic* may succeed if the former is insufficient, but the *arsenite of copper* in the third decimal trituration is the most effective medicine.

The pains and other symptoms simulating cerebro-spinal fever call for *bryonia*, *actea*, *agaricus*, and *cuprum acet*.

For the anæmia and depressed tissue nutritions the *iodides of arsenic and of iron*, and *ferrum phosphoricum*, have been beneficial. If the nervous tissues have suffered most, *zinc phosphide* has proven a useful "tonic" to the cerebral tissues; and for general neurasthenic symptoms, the *arsenate of quinine* and the *arsenate of strychnine*. Each of these medicines is best administered in doses of the  $\frac{1}{100}$ th gr. (second decimal trituration in the form of a tablet triturate) several times daily.

No matter what the character of the sequelæ, it must be remembered that much more than medicine is required for the relief of many cases. The patient should be kept under observation during convalescence on account of the danger of the development of phthisis and other serious consequences.

## YELLOW FEVER.

**Nomenclature.**—This affection has received a great variety of names, but as the one heading this article has been generally accepted, it is entirely unnecessary to give those which are now obsolete. The name is based on a prominent clinical feature, *i.e.*, the yellow color of the skin, which is generally present.

**Definition.**—Yellow fever is an acute specific infectious disease, endemic in tropical countries, from which it extends at intervals to cooler regions. Under favoring conditions it assumes the form of an epidemic. Typical cases are characterized by fever, yellowness of the skin, albuminuria, and hæmorrhages from the mucous membranes, especially that of the stomach.

**History.**—Guiteras, who has studied this disease with commendable zeal, describes three zones within which yellow fever prevails: *First*, the *focal zone*, which includes the “ports of Havana, Vera Cruz, Matanzas, Rio Janeiro, and other important seaports of inter-tropical America, perhaps also a portion of the Atlantic coast of Africa. In these parts the disease is never entirely absent, and regularly spreads every year with the advent of warm weather and invades the inhabitants who are not protected by a previous attack, with few exceptions.”

*Second.* “The *perifocal zone*, or region of periodic epidemics.” To this zone belongs the “majority of the ports of the tropical Atlantic in America and Africa, and a smaller number on the Pacific coast of the American Continent.”

*Third.* The *zone of accidental epidemics*, which “includes those places of the temperate zone or of high altitude, where the disease is occasionally imported, and will prevail for one season only.” The boundaries of this latter zone cannot be positively set, but the disease may become epidemic anywhere between the parallels of 45° north latitude, and 35° south latitude. “The epidemics of New York, Philadelphia, the Mississippi Valley, Florida, Spain, Montevideo, Cuzco, etc., are examples of the third group.” It is evident that the region of greatest prevalence is the West Indies and the seaports upon the Gulf of Mexico. The free commercial intercourse existing between these ports and the cities of the United States further north has led to importation of the disease at intervals, and the establishment of extensive epidemics. It is especially the cities of the Southern States and of the Atlantic seaboard further north which have suffered. Extensive epidemics occurred in 1793, 1797, 1798, 1799, 1802, 1803, 1805, 1853, 1867, 1873, 1878. The epidemic of 1793 was

especially extensive and very fatal, the mortality in Philadelphia alone being over four thousand with a population of hardly forty thousand." About the same number of persons have perished in this city during the other epidemics of yellow fever of which we have accounts. The disease has not yet been epidemic in the British Isles or France, although their seaport towns have been invaded. The yellow fever epidemic of 1793 in Philadelphia made its first appearance in Kensington, and was attributed to "a heap of putrid coffee and some piles of stinking hides that had long cumbered one of the wharves near Mulberry Street. Along the best thoroughfares the mud and filth were very deep. On the vacant lots and under the very windows of some of the most frequented inns, the carcasses of horses lay rotting in the summer sun." The onset of the disease was treated with bleeding, starving and purging; one case is recorded where seventy-two ounces of blood were let in as many hours.

Fires were kept burning on the street corners, but the physicians announced "that they had little faith in them as purifiers of the air, and much in the burning of gunpowder." Instantly every body who had a gun, loaded and fired it from morning to night. This was so noisy "that the doctors ordered it stopped. Then the people began to burn nitre instead. One day tobacco was thought to be a good preventive, and the dealers could not supply the demand; on another, garlic was recommended, which some chewed and others put in their shoes; some went about with huge bunches protruding from their coat pockets. Then it was discovered that camphor was a disinfectant, and in a little while everyone had a great bag of it strung around his neck. But no medicine was so popular as the vinegar of the four thieves." As one sallied forth on the street he must have presented a ludicrous sight "with a piece of tarred rope in either hand, a sponge wet with camphor at his nose, and in his pocket a handkerchief saturated with the latest preventive of which he had heard."

A hospital was established at Bush Hill with Stephen Girard as superintendent. No nurses could be had but prostitutes, who rioted on the dainties sent to the sick. The mortality here was frightful, and many ill with the disease rushed out of the city and "perished in haystacks and ditches." Little children, hungry, orphaned and homeless, wandered starving in the streets. For this fragmentary account we are indebted to McMaster's "History of the People of the United States," Vol. IV, pp. 125-134.

**Etiology.**—Historical data seem to establish the fact that yellow fever in the United States first appears, as in other countries, in seaport towns, and from which it extends inland along the lines of commerce. This extension is a slow one. The introduction of the disease into a seaport is almost invariably by some vessels carrying infected materials or patients ill of the disease. The first case in the community generally



makes its appearance about ten days or two weeks after the arrival of the vessel. The infectious agent, whatever it may be, may be carried by clothing or other formites. The white race and temporary residents in a community are most susceptible to the contagion. Negroes are less frequently attacked than whites. Children were formerly regarded as seldom suffering from yellow fever, but Guiteras's researches have shown this not to be the case. He shows that Creole infants are frequently attacked, the true nature of their illness fails of recognition, and they thus maintain focal endemicity of yellow fever. One attack of yellow fever, however mild, almost certainly preserves the patient from danger of subsequent ones. Long residence in the infected locality is partially protective. Men are more frequently attacked than women, probably because they are more exposed.

Unfavorable conditions are a residence at the seacoast, warm weather, especially during the months of June, July and August, and everything favoring depression of the general health, *i. e.*, overcrowding, unhygienic and unsanitary conditions generally. On the other hand, the spread of the infectious agent is prevented by certain singular agencies. Thus a stream will mark the line of an epidemic; in other cases a high wall will have a similar influence. The poison evidently keeps very close to the ground, as the majority of cases occur at less than an altitude of 750 feet above the sea-level.

The specific cause of yellow fever has not been demonstrated, although as to its bacterial nature there can be but little doubt. Freire, it is true, has discovered a micro-organism which he regards as the essential cause of the disease, but its specific relation to yellow fever has been doubted by many observers, and especially by Sternberg. Our knowledge of the acute infectious diseases in general justifies us only in believing that in yellow fever, as in cholera, typhoid and relapsing fevers, we have to do with a micro-organism as the specific agent. It seems quite certain, also, that this organism may develop outside of the human body. Sternberg's researches lead him to the opinion that it is found especially in the fæces, and that out of the body it is capable of cultivation in accumulations of fæces and organic matter of animal origin, when climatic conditions are favorable to its growth. There is abundant evidence to show that the yellow fever poison is portable, that it retains its vitality for a great length of time, attaching itself to holds of vessels, clothing, etc., that it is capable of a high degree of concentration in unventilated and filthy quarters, and that the progress of the disease is arrested by cold weather. It can be taken into the body either by the respiratory tract or the alimentary canal.

**Pathology and Morbid Anatomy.**—An icteric color of the skin, decomposition of the blood, fatty degeneration of the liver, acute catarrh of the gastro-intestinal mucous membrane, with perhaps hæmorrhagic



spots, and an accumulation of altered blood in the stomach similar to the "black vomit," diffuse nephritis, pulmonary infarctions, and extravasations of blood in the various tissues and organs, are the most important pathological changes found in yellow fever. The blood changes are most important, as many of the conditions enumerated are the direct result of the alterations in this fluid. The departures from the normal are in kind the same as occur in the acute infectious fevers generally, but are of a higher grade of intensity; especially is this true of the corpuscular changes. Many of these bodies are destroyed, others shrunk or serrated. Recent observations indicate that the destruction of blood corpuscles is not as great as was at one time taught. The blood is much darker than normal, and its coagulability diminished. It contains free hæmoglobin. That the color of the blood and the tissues is due to blood changes and not to the presence of bile has again been recently satisfactorily determined by the chemical analysis of a French writer, Cunisset. The most pronounced and important pathological changes are to be found in the abdominal organs, especially in the liver. This gland is of a pale yellowish or brownish color, the coloring being general or in patches. There may be extravasations of blood upon its surface, although the organ is usually anæmic, except in persons dying early, in which case hyperæmia may be found. The tissue is dryer than normal, and breaks down readily. The microscope exhibits liver cells in varying states of fatty change, as well as areas of necrosis. The kidneys are not often much altered in appearance, even when they are the seat of parenchymatous nephritis, as they almost constantly are. Hæmorrhagic foci are common. The spleen is somewhat enlarged, softened, and darker than normal. The lungs contain infarctions, which are sometimes quite numerous. The pleuræ may be covered with ecchymotic spots and a moderate quantity of bloody fluid may occupy the pleural sac. The stomach as well as the œsophagus and intestinal tube are invariably the seat of a slight catarrhal inflammation. The stomach presents foci of hyperæmia of an irregular outline and a radiating periphery. There may be hæmorrhagic foci. The contained dark fluid consists essentially of blood darkened by the action of the gastric juice. Crevaux states that the cells lining the gastric follicles undergo fatty degeneration. He considers this the most important lesion found in the stomach.

The brain and spinal cord may be congested, and hæmorrhagic points are sometimes found in the meninges.

Both for study and clinical observation it is convenient to divide yellow fever into three stages, as follows: (1) The initial chill, followed by febrile reaction; (2) Period of remission, or stage of calm; (3) Uræmic stage, or exacerbation, or collapse.

**Clinical Course.**—It is not common for prodromic symptoms to precede the onset of yellow fever. If present, there is nothing peculiar

in their character deserving mention. The attack is usually ushered in by a rigor, although the chill may be absent even in grave cases. Its length and intensity bear some relationship to the violence of the attack. The rise of temperature is rapid: During the chill, and often aggravated during the fever, are nausea, vomiting, frontal headache, severe pains in the back and limbs, a flushed face, shining and staring eyes, and, perhaps, pain in the eyeballs, with photophobia.

The temperature in yellow fever is not generally a very high one. Temperatures of from  $108^{\circ}$  to  $110^{\circ}$  F. have been reported, but they are phenomenal. High post-mortem temperatures have been recorded. The usual range is about as follows: The temperature rises rapidly after the chill, attaining its acme in a few hours; or, in severe cases, the attainment of the highest point is not accomplished until the close of the second or even of the third day. A rise during this time to  $104.5^{\circ}$  and  $106^{\circ}$  F. is not uncommon. From this often speedily-attained acme there is a gradual fall to the normal in from four to ten days; usually, in well-developed cases, about the eighth or ninth day. Often the temperature falls suddenly to nearly normal, or even below normal, upon the fourth day, thus closing the period of initial fever. This marked remission lasts from a few hours to two or three days, and is followed by a second rise, called the reactionary fever. The temperature range in this second fever stage is rather irregular, showing remissions and exacerbations, and it is of uncertain duration.

The pulse may be but slightly accelerated in mild cases, and even in severe attacks, with a high temperature, it seldom rises above 100 to 110. Falligant, however, in his article in Arndt's "System of Medicine," speaks of the pulse as attaining 130 or 140, "according to the age of the patient." With the progress of the attack (according to Faget) the pulse loses in tension and frequency, even though the temperature remains high. This he looks upon as a valuable diagnostic sign. The highly depressing and degenerating influence of the yellow fever poison upon the heart is indicated by the frequency, during the second stage, of a slow, compressible pulse, termed by some a "gaseous pulse." Reductions to 50, 40, or even less, are occasionally noted. The surface of the body may be either hot or dry, moist, or bathed in perspiration during the first stage. With the remission the skin generally becomes cooler, and a gentle perspiration appears. If a fatal issue occurs at this time, the body may be bathed in a clammy sweat. Rarely the skin is hot from first to last, such cases usually ending fatally.

By the second day the patient emits a peculiar odor, which has been variously described as "sickly," "fishy," "corpse-like," etc.

The yellowish tint of the skin, which has given rise to the generally accepted name, is present in a varying percentage of cases in different epidemics. The color varies from the faintest yellowish tint to a saffron

or mahogany. It is gradually developed during the febrile stage, being generally noticeable by the third day, and is first visible in the sclera. The prevailing opinion, as heretofore noticed, is that this jaundiced appearance of the skin is of a hæmatogenous character. Ferand and Gubler have recently advocated the dual character of this icterus. The first coloring, which appears late in the initial fever paroxysm, is stated by them to be due to blood changes only. A second form, appearing late in the stage of remission, or even during convalescence, is due to the presence of bile pigments; and it is this form which causes the occasional deep orange or mahogany color. This hepatogenous jaundice cannot be considered as an essential feature, but rather as a complication of yellow fever.

**STOMACH, MUCOUS MEMBRANES, ETC.** Nausea and vomiting usually follow closely upon the heels of the chill. The vomited matters at first consist of the ordinary gastric contents, then of bile-stained fluids. Owing to ammoniacal decomposition the vomited substances are of alkaline reaction. In serious cases they become gradually transformed into the so-called black vomit, which derives its characteristics from the admixture of blood altered by the stomach fluids. This symptom occurs in from one-quarter to one-third of the cases of yellow fever, and is of ill omen, comparatively few recovering after its appearance. While it may appear early in the disease, it is seldom seen until near its close, viz., during the last forty-eight hours. This symptom is not at all peculiar to yellow fever, since it occurs in any disease in which slow hæmorrhage into the stomach takes place.

Even when the "black vomit" has not appeared during life, it may be discovered in the stomach after death. The epigastrium is usually tender, and the patient complains of distress or decided pain. There are also anorexia, thirst, and constipation.

Hæmorrhages may occur from almost any portion of the mucous membranes. Epistaxis, hæmorrhage from the tongue, gums and lips, are common, less frequently from the intestines, bladder, uterus, eyes and ears.

**URINE.** As in febrile diseases generally, the urine is diminished during the early fever, and a little albumin may be present. Later, however, there takes place a marked decrease in the quantity of the urine, and this is not to be explained in the manner usual to the infectious fevers. Partial or complete suppression is a prominent characteristic of fatal cases, resulting in uræmic manifestations. Quite extensive albuminuria is almost constantly found, at least, by the time of the stage of depression; a scanty secretion of highly albuminous urine at this time is a most unfavorable indication. The elimination of urea and uric acid is diminished. The reaction of the urine is generally markedly acid.



In cases of little severity, the mind is not at all disturbed. Delirium is uncommon, even in cases passing to a fatal termination. Somnolence, passing gradually into coma, is more frequent. Convulsions occur rarely, and usually just prior to death. During the early stage the patient is restless, complaining of pain and sleeping but little, and what sleep he gets may be disturbed by distressing dreams. A certain amount of anxiety and mental activity are often present.

**Diagnosis.**—The symptomatology of yellow fever is so fully defined that it would seem almost impossible to confound the disease with any other. Yet mistakes seem to occur. The most common is believed to be that of regarding many cases occurring, especially in children, as examples of malarial fevers. The failure to recognize these forms is a source of great danger to the community in which they occur. The important diagnostic symptoms are the peculiar face of the patient, the temperature curve, the relation between temperature and pulse-rate, and the discoloration of the skin. The appearance of the face has been variously described as besotted, or resembling the face of measles prior to the outbreak of the rash. The temperature curve and the pulse-temperature ratio have already been described.

Bacteriological examination of the blood affords a ready method of differentiation from malarial fevers. In the latter the micro-organism of Laveran is always found.

Much stress has been laid on albuminuria as a diagnostic sign. This symptom is unreliable for diagnostic purposes, as it may be present in any of the conditions which may simulate yellow fever.

**Prognosis.**—In favorable cases the reactionary fever quickly gives way and the patient passes into a tedious convalescence, marked by feebleness, especially of the digestive organs, demanding great care in the management of the diet. In unfavorable cases vomiting persists, the urine is scanty and highly albuminous, and serious nervous symptoms appear. A high temperature in the early days, the rapid supervention of jaundice, "black vomit" and epigastric distress, as well as of suppression of urine, and a well-marked albuminuria, are all unfavorable. While the "black vomit" is a most serious symptom, cases presenting it not infrequently recover. Death may occur within the first twenty-four hours of the disease from the intensity of the toxæmia. More frequently, however, it does not occur for five or six days. Occurring in subjects who have led hygienic lives, the prognosis is far better than in others. While serious and even fatal attacks are noted in total abstainers, still the latter have immeasurably better chances of life than the moderate drinker or the drunkard.

The mortality rate differs very much in different epidemics. It is much less in cities and towns in which the disease is epidemic, or frequently epidemic. Rates as low as 7 per cent. have been reported from



such places. Great epidemics in unfrequented cities have given as high as 80 per cent. of deaths. Between these extremes one can make any statement and find evidence to support it.

**Treatment.**—The best prophylactic measure is removal from the infected district. Preventive inoculations have been strongly urged by Freire and his colleagues. Their value does not seem to have as yet been thoroughly attested by the majority of physicians. The more recently recommended serum therapy is likewise wanting in favor by the vast majority of those competent to give a decision. Medicines alleged to act as preventives have not found favor. People who must remain should do all in their power to maintain their general health at the highest possible standard. Regular hours, feeding and rest are important. Some observers think there is more danger from the night air. A rigid quarantine, including not only the patient, but all the articles used in and about the sick-room, should be established.

With the beginning of the illness, the patient should be put to bed, and not allowed to leave it for any purpose whatever until convalescence has been thoroughly established. Even exertion in bed must be prohibited. Violation of this rule, even in the early stage of convalescence, has often been followed by serious results. If circumstances compel the removal of the patient after the beginning of the disease, his recovery is greatly endangered thereby. An aggregation of patients in one room is to be avoided if possible. All excreta should be disinfected before disposing of them. The patient must be placed in the charge of a well-qualified nurse. There is no disease in which it is of greater importance to institute efficient treatment early. Every attention, however slight, adds much to the patient's chances of recovery.

**ALIMENTATION.** Those having the largest experience advise the discontinuance of food of every character during the first days, *i. e.*, until the remission, or period of "calm." Then they suggest that the patient begin with carefully selected liquids, which are to be given in teaspoonful doses only. The effect of the first dose must be most carefully observed before repeating it. Chicken-water, iced milk, milk and barley-water, etc., have been suggested by different observers as especially suitable for the first trial. This course is not well borne, however, by children, or cases of a typhoid character, or by those suffering from hæmorrhages or marked prostration. Rectal alimentation can generally be carried out successfully in this affection, and has doubtless saved many lives. Cool drinks are admissible. Carbonated water seems especially acceptable and useful. Shaved ice is allowable. Wine and beer are permitted by some physicians at all stages, being considered by them valuable, therapeutically. All drinks and food should be administered to the patient without raising him from his recumbent posture.

**STIMULANTS.** As a rule, stimulants are not called for until after the

fourth day. The indications for their use are the same as for the use of alcohol in any of the acute prostrating diseases. At first the dose should be small, and frequently repeated. Later, however, larger doses to support a failing circulation may become a necessity. The best form of stimulant is not conceded to be the same by all authorities. Most physicians recommend brandy, others champagne, and Blair lays stress upon the superiority of Rhenish wine over all other liquors. He often gives it in very large quantities.

Emetics, purgatives, bleeding, and many drugs and "methods of treatment" have been employed by the old school, but the present view entertained is well expressed by Sternberg, who writes, "a majority of the physicians in those parts of the world where yellow fever prevails, who have had an extended experience, agree that active medication is injurious." Making allowance for undue laudation and absence of sufficient statistics from hospitals, I am convinced of the great superiority of the homœopathic method in the treatment of yellow fever. Our experience has been carefully detailed by a number of physicians, but especially by Drs. Holcombe, Morse, and Falligant. The experience of these men has been so great, and their results so uniform in degree, that I shall draw my suggestions as to treatment very largely from their writings.

In the mild uncomplicated cases, it was Falligant's custom to administer *aconite* and *belladonna* in alternation. The mother-tinctures of both remedies were employed. *Camphor* yields good results in cases characterized by chill, even when the condition is sufficiently severe to produce collapsic symptoms. It should be given in frequently-repeated doses in order to secure the best possible results. *Veratrum album* and *aconite* may also be thought of in the same condition. In all such cases the use of a hot foot-bath furnishes a valuable adjuvant. *Ipecacuanha* should be given when the nausea and vomiting become prominent symptoms. *Byronia* should also be considered in this connection.

For the late vomiting, Holcombe places reliance upon *argentum nitricum*. Other observers praise *lachesis* and *crotalus* for this symptom; the latter remedy being especially preferable if the "black vomit" is present. *Cadmium sulphuricum* is symptomatically suggested by the latter condition. For the flatulency and its attendant symptoms, *nux vomica* is to be thought of. For the subsequent biliary symptoms and weak stomach, *nux vomica* and *mercurius dulcis* in  $\frac{1}{200}$ th of a grain doses (Falligant). For the cerebral symptoms, *belladonna*, *byronia*, *hyoscyamus* and *opium*. The vomiting of the second stage is said to be checked by *sulphuric acid* or *arsenicum*. For cystic hæmorrhage, *cantharis*, *belladonna* and *arsenicum*. For uterine hæmorrhage, *secale* and *arsenicum*. For the convulsive symptoms, *belladonna*, *hyoscyamus*, *opium* and *stramonium*. For the nervous and typhoid forms of the disease, *byronia*, *arsenicum*, *rhus*, *china*, *nux vomica*, *carbo veg.* and *mercurius solubilis*. *Cantharis* is regarded

by Holcombe as the most efficient remedy for the suppression of urine. It is especially indicated when there are burning pains in the stomach, slow pulse, hæmorrhages from the stomach and intestines, and cold sweat on the hands and feet. The hot bath must be remembered as a valuable adjuvant when uræmia threatens. *Cuprum* has checked the vomiting of blood after arsenicum has failed. The *arsenite of copper* is here worthy of attention. *Platinum* has been recommended very highly by Holcombe for the intense wakefulness; especially is it called for by a discharge of dark, almost black, menstrual blood. *Glonoin* has proven useful in some cases in which meningeal inflammation threatened.

The snake poisons, notably *lachesis* and *crotalus*, have long enjoyed an enviable reputation in the treatment of yellow fever. Holcombe and Neidhard especially have asserted them to be essential remedies in this disease. In view of their recommendations, it is interesting to know that in the endeavor to secure a prophylactic remedy, the poison of the *crotalus horridus* has been administered extensively in New Orleans and Cuba as an inoculation remedy, and, it is claimed, with considerable success. These inoculations have at least provided us with valuable provings of the poisons. *Lachesis* is called for in all stages of the disease, "but particularly after the abuse of quinine and mercury." Holcombe recommended it in the earliest stage, and *crotalus* in the second, when jaundice, hæmorrhages and great enfeeblement are present. Neidhard and Hughes corroborate these observations. *Tartar emetic* is recommended for the excessive vomiting, sinking feeling at the epigastrium, as if one would die; prostration with cold sweat; feeble pulse; drowsiness. *Carbo veg.* was suggested by Hering as corresponding better than any other medicine to the totality of the conditions found in yellow fever. His observations have been confirmed by a number of physicians. The hæmorrhages, the wretched appearance, the feeble circulation, flatulence, burning in the epigastrium, offensiveness of the discharges, restlessness, cyanosis, coldness of the surface, and ecchymoses, are suggestive of the possible value of *carbo veg.* *Phosphorus*, owing to its remarkable influence upon the liver, inducing fatty degeneration of that organ and producing jaundice, albuminuria and hæmorrhages, has been suggested as a yellow fever remedy. There is, however, but little evidence to show its successful use.

## DYSENTERY.

**Definition.**—An acceptable definition of the term “dysentery” can hardly be advanced in the present state of our knowledge. There can be no doubt that even at the present day, as in ages past, quite a variety of morbid conditions presenting analogous clinical phenomena, but dependent upon entirely different causes, have been classed under this name. Some of these are undoubtedly sporadic, being producible by certain external agencies, which, in the case of the epidemic or infectious cases, act as predisposing causes. Dysentery must then be regarded as but a generic term, including all cases characterized clinically by frequent discharges from the bowels, at first of a simple diarrhoeal nature, but later consisting of mucus, blood and pus, and accompanied by severe rectal tenesmus and abdominal pain, and dependent upon inflammatory and usually ulcerative and pseudo-membranous changes in the large intestines.

**History.**—The history of very few, if of any diseases can be traced to as remote ages as can that of dysentery. This is probably due to the fact that this affection has ever been the scourge of armies, thus bringing it forcibly to the attention of the ancient historians, who, after all, had but little to do other than to chronicle the fortunes, misfortunes and intrigues of war. Herodotus mentioned an epidemic of dysentery occurring in the Persian army under Xerxes while marching through Thessaly. Hippocrates himself described the condition under the very name it now bears; indeed, it is more than probable that he is responsible for that name. While his description of the disease was a most excellent one, he included quite a number of morbid conditions which were subsequently eliminated by others. Aræti<sup>us</sup>, in A. D. 50, gave a very accurate description of the clinical phenomena of dysentery, together with a most admirable account of the pathological changes present. Since those ancient days, the advances respecting our knowledge of dysentery have been mainly in the direction of etiology and treatment; and yet we are still obliged to confess that concerning the origin of few diseases do such confused ideas and theories prevail. The presentation and rejection of theory after theory, concerning the advent of dysentery epidemics, furnishes a most interesting part of the history of medicine.

**Etiology.**—A proper understanding of the etiological factors of dysentery compels us at once to recognize two classes of cases, the sporadic and the epidemic, the exciting causes of the former acting as the predisposing in the latter. This view is, of course, denied by some good authori-



ties who firmly believe that dysentery is an infectious disease, and that it can never arise independently of the specific agent, germ or miasm though it be. Eichhorst, for example, says that it never occurs autochthonously, and explains the apparently idiopathic cases by suggesting that they result from exposure to mild cases which escape recognition by physicians and others.

Climate exerts an influence greater probably in the production of dysentery than in any other disease. It is especially liable to appear endemically in tropical countries, notably the East Indies. The temperate zones bordering on the tropics are likewise liable to its ravages. It is, indeed, in all these places a far more active agent of death than the more dreaded cholera. At 40° latitude and beyond, dysentery ceases to occur as an epidemic disease. So commonly is this disease present in tropical climates that it has been stated there that "all cases of fatal acute or chronic disease finally perish with it." In non-tropical countries in which dysentery gains a foothold, it does so during the hot periods. Apparent as is heat as a cause of dysentery, it is not a necessary factor, for epidemics have been observed in mid-winter and in the cold climates of Russia, Sweden, and Canada. On the other hand, exceptionally hot seasons have prevailed, in which dysentery was notable for its absence.

It has been suggested that the prevalence of dysentery in tropical climates is the result of the combination of heat, moisture, and changeable temperature. The heat and moisture undoubtedly tend to promote vegetable decomposition, a prolific cause of vitiation of the atmosphere. Annesley, writing in 1828, observed that the disease was much more liable to break out in regiments stationed along rivers and lowlands, where the emanations from decaying vegetable and animal matter were especially great. In Egypt, epidemics of the disease are liable to occur after the overflow of the Nile. The removal of camps, in which dysentery is rampant, to higher and dryer localities has often been followed by stamping out of the disease. Then again, as if to impress confusion into the study of the etiology of dysentery, we find epidemics—exceptional ones, it is true—occurring in climates and stations remarkable for their dryness. Annesley sums up the relation between dysentery and moisture as follows: "All situations which furnish exhalations from the decay of animal or vegetable productions under the operation of a moist and hot state of the atmosphere, will always occasion dysentery in the predisposed subject—circumstances which, with other causes, combine to generate the disease."

Sudden changes in temperature, especially the succession of hot days by cold nights, appear to cause numerous cases of sporadic dysentery in our own climate, and predispose to epidemics of the disease. Sleeping on damp ground, exposure of the abdomen during sleep, the neglect to wear proper woolen underclothing, have each been productive of dysen-

tery. Interesting examples of the influence of exposure in the causation of epidemics have been cited as occurring in armies after a heavy rainfall and lowering of the temperature; and on shipboard after all clothing and bedding had been thoroughly saturated.

Nervous influences sometimes play an important part. In military life, the disease has been noted as especially severe among the prisoners of war. Whether this is due to the attendant mental depression or to the privation to which the vanquished are subjected by the victors, is uncertain, both are probably influential.

Malaria has been said to occasion dysentery. It is true that the disease does often occur in the midst of malarial surroundings. But it may occur in regions in which malaria is unknown. Even in malarial districts, dysentery and intermittent fevers may occur independently of each other—one prevailing at times when the other is noteworthy for its absence.

Dietetic transgression are undoubtedly etiological factors. The eating of unripe or decomposing fruit or drinking of contaminated water, the excessive use of salt meat, and malt liquors have been responsible for sporadic cases of the disorder. General errors in diet, by producing dyspepsia, lower the systemic and local resistance and thus favor the production of dysentery.

In many instances the character of the soil has apparently great influence in the production of the disease. Countries in which the soil is of a character suitable for the propagation of malaria, are as a rule the ones in which dysentery is most prevalent. From this fact, however, one must not generalize too much, and assume that malaria and dysentery arise from a common miasm. The conclusion should be rather that of Huebner, viz., that the cause of epidemic dysentery is “a miasma which is developed under the influence of a tropical climate or one resembling a tropical climate, in a soil of a certain moist and perhaps swampy character, in an analogous way with malarial poisoning.”

Opinions differ greatly as to the contagiousness of dysentery. It is almost certain, however, that the disease is slightly contagious, some few instances in which it has been transmitted by third parties having been reported. This transmission seems to be by means of the stools almost entirely. The stools are especially infectious when a large quantity of fecal matter from dysenteric patients is collected, thus concentrating the poison as it were. Carelessness in the management of the excreta certainly favors the dissemination of the disease. If the stools be permitted to soak in the ground, the latter can certainly act as a culture-bed, and in this way it readily poisons both air and water. The spread of dysentery from water-closets is now apparently a well-attested fact.

Some German authorities have laid considerable stress on obstinate constipation as a cause of dysentery. The retention of large scybalous

masses in the bowel must occasionally produce a catarrhal inflammation which, under the influence of proper predisposing influences, can be readily supposed to favor an attack of the disease.

**Pathology and Morbid Anatomy.**—From an anatomical standpoint cases of dysentery may be divided into the catarrhal, amœbic or tropical, and diphtheritic.

(1) ACUTE CATARRHAL DYSENTERY. The lesions are generally found in the rectum and lower bowel, but may extend into the ileum. In short and severe attacks marked changes are often found throughout a greater or lesser portion of the small intestines. The small intestines are distended with gas, the large intestines, with the exception of the distended transverse colon, are empty.

In the early stage the solitary glands are enlarged with increased vascularization of the surrounding mucous membrane, accompanied by softening and swelling. The solitary glands project slightly and are about the size of a mustard seed. As the disease advances the glands become capped with necrotic tissue which on sloughing leaves a small circular ulcer with rounded edges. Again several glands with the intervening mucous membrane may slough simultaneously, leaving a large, irregular ulcer with ragged edges, many of the larger ulcers having a transverse direction.

The depth of the ulcers varies greatly. One or all coats of intestines may be involved, perforation following. Resolution occurs by the edges becoming rounded and adhering to the base, the cavity filling with lymph exudate which becomes organized. New glands do not form in the cicatrices. Healing may or may not be attended with contraction. A slight puckering generally follows the contraction, or severe stricture by the continued contraction of the cicatrices. The mucous membrane of the digestive tract above the lesions is inflamed. The stomach is usually distended, but it may be contracted, and its mucous membrane thickened and softened. Other complications are rare.

(2) AMŒBIC OR TROPICAL DYSENTERY. This variety has been best described by Osler, indeed as most of our knowledge of this variety comes from the pathologists of the Johns Hopkins school, I shall therefore take the liberty of presenting Osler's description of the post-mortem appearances in his own words:

"The lesions are found in the large intestines, sometimes in the lower portion of the ileum. Abscess of the liver is a common sequence. Perforation into the right lung is not infrequent.

"*Intestines.* The lesions consist of ulceration produced by preceding infiltration, general or local, of the submucosa, the general infiltration being due to an œdematous condition, the local to multiplication of the fixed cells of the tissue. In the earliest stage these local infiltrations appear as hemispherical elevations above the general level of the mucosa.



The mucous membrane over these soon becomes necrotic and is cast off, exposing the infiltrated submucous tissue as a grayish-yellow gelatinous mass, which at first forms the floor of the ulcers, but is subsequently cast off as a slough.

“The individual ulcers are round, oval or irregular, with infiltrated undermined edges. The visible aperture is often small compared to the loss of tissue beneath it, the ulcers undermining the mucosa, coalescing, and forming sinuous tracts bridged over by apparently normal mucous membrane. According to the stage at which the lesions are observed, the floor of the ulcer may be formed by the submucous, the muscular or the serous coat of the intestine. The ulceration may affect the whole or some portion of the large intestine, particularly the cæcum, the hepatic and sigmoid flexures, and the rectum. In severe cases the whole of the intestine is much thickened and riddled with ulcers, with only here and there islands of intact mucous membrane.

“The disease advances by progressive infiltration of the connective tissue layers of the intestine, which produces necrosis of the overlying structures. Thus in severe cases there may be different parts of the bowel sloughing *en masse* of the mucosa or submucosa or of the muscularis, and the same process is observed, but not so conspicuously, in the less severe forms.

“In some cases a secondary diphtheritic inflammation complicates the original lesions.

“Healing takes place by the gradual formation of fibrous tissue in the floor and at the edges of the ulcers, which may ultimately result in partial and irregular strictures of the bowel.

“Microscopical examination shows a notable absence of the products of purulent inflammation. In the infiltrated tissues polynuclear leucocytes are seldom found, and never constitute purulent collections. On the other hand, there is proliferation of the fixed connective tissue cells. Amœba are found more or less abundantly in the tissues at the base of and around the ulcers, in the lymphatic spaces, and occasionally in the bloodvessels.

“The lesions in the liver are of two kinds: firstly, local necrosis of the parenchyma, scattered throughout the liver, and possibly due to the action of chemical products of the amœba; and secondly, abscesses. These may be single or multiple. When single they are generally in the right lobe, either towards the convex surface near its diaphragmatic attachment, or on the concave surface in proximity to the bowel. Multiple abscesses are small and generally superficial. In an early stage the abscesses are grayish-yellow with sharply defined contours, and contain a spongy necrotic material, with more or less fluid in its interstices. The larger abscesses have ragged necrotic walls, and contain a more or less viscid, greenish-yellow purulent material mixed with blood and shreds



of liver tissue. The older abscesses have fibrous walls of a dense, almost cartilaginous consistence or toughness. A section of the abscess wall shows an inner necrotic zone, a middle zone in which there is great proliferation of the connective tissue cells and compression and atrophy of the liver cells, and an outer zone of intense hyperæmia. There is the same absence of purulent inflammation as in the intestine, except in those cases in which a secondary infection with pyogenic organisms has taken place. The material from the abscess cavity shows chiefly fatty and granular detritus, few cellular elements, and more or less numerous amœbæ. These organisms are also found in the abscess walls, chiefly in the inner necrotic zone. Cultures are usually sterile. Lesions in the lungs are seen when an abscess of the liver—as so frequently happens—points towards the diaphragm and extends by continuity through it into the lower lobe of the right lung. The gross and microscopical appearances are similar to those of the liver.”

(3) DIPHTHERITIC DYSENTERY. The lesion is found in the colon and the first two or three feet of the ileum. Changes higher up are seldom observed; the disease being quite as severe in the ileum as in the colon. The area of greatest inflammation is at the ileo-cæcal valve, next in severity the sigmoid flexure and rectum. The disease is characterized by areas of necrosis of the mucous membrane, which on sloughing leaves an ulcer.

In severe cases the colon is distended, the mucous membrane thickened, infiltrated and stiff, with no traces of glandular tissue. Peyer's patches and the solitary glands are indistinct or entirely lost. The necrosis may involve only the upper layer of the mucous membrane, or all the coats of the intestines down to the serous are involved. The pseudo-membrane may be in small patches, or large areas may slough, while in cases of long duration the slough often comes away in large tubular pieces. In mild cases it is rare to find large areas of pseudo-membrane that can be detached. These are grayish green, and the mucous membrane between is usually of an intense red color, rough and granular.

In secondary diphtheritic dysentery there is usually only a thin, superficial, localized infiltration, generally in the ridges and folds of the mucosa, of the colon and ileum. In severe cases the entire mucosa may be involved and necrotic, and of a rough granular appearance. It occurs at the end of acute and chronic diseases, as chronic heart disease, Bright's disease, cachectic states generally, and often pneumonia.

CHRONIC DYSENTERY. This usually follows an acute attack, or the amœbic variety. But some cases are chronic from the start. The anatomical changes are various. The intestines are generally thicker and firmer than normal, of a slate color, gray or blackish, or they may be greatly distended until they are as thin as parchment. There may be no ulcerations, the villi are indistinct or missing, and the mucosa looks

rough, irregular and puckered. In fact, the lesions may be entirely overlooked.

As a rule ulcers are found, often large, many in the course of healing, and are deeply pigmented. The muscular and mucous coats thickened, causing narrowing of the bowels, but strictures are rare. The mesenteric glands are enlarged and pale.

**Clinical Course.**—In the epidemic disease there is a stage of incubation ranging from three to eight days. The prodromal symptoms are of the usually indefinite character, or may be entirely absent. In the latter case the disease may or may not be ushered in by a chill. Diarrhœa appears, at first without, and later with pain, which becomes more severe with the advance of the disease. In the course of one or two days the stools change in their appearance and assume the dysenteric features, that is to say, they lose their fæcal character and consist almost solely of blood, mucus, pus and detritus. They are now accompanied by severe rectal tenesmus and abdominal pains, these latter sources of suffering continuing with varying severity during the intervals between the stools. The temperature is usually above normal, but rarely if ever higher than 102° or 103° F.

**Analysis of the Symptoms.**—Considering first the stools, we find these in the first days of the disease differing in no particular from those of an ordinary diarrhœa, unless perhaps they are attended with more or less tenesmus. They then lose their fæcal characteristics, and contain blood and mucus, and have been described as gelatinous. Generally they are nearly odorless, excepting in the diphtheritic variety, when the presence of sloughs gives them a highly gangrenous odor. They are always small in quantity; indeed so small that, notwithstanding their great frequency, the aggregate quantity passed during the twenty-four hours does not exceed thirty-six ounces, and may be much less. The number of stools *per diem* varies according to the severity of the case, ranging from but a few to as many as two hundred.

Huebner has described the appearances of the different varieties of dysenteric stools, and gives the following: (1) The mucous or muco-sanguineous stool, consisting of a slightly yellowish, glassy, quivering mass, lying in the vessel in balls or clumps. (2) The sanguineo-purulent stool (*lotio carnea*), consisting of a yellowish or reddish fluid, in which float numerous soft yellow, red or reddish lumps, which bear a certain resemblance to raw minced meat. These lumps consist of a thick mucus network, in which red blood corpuscles are entangled. (3) The pure bloody stool. This is the result of a superficial hæmorrhage in the beginning or of the erosion of bloodvessels later. (4) Discharge of pure pus, which occurs only in the later stages. (5) Gangrenous stools, which are brownish-red or black in color. Their odor, as already stated, is putrid, and they indicate gangrene of the intestinal mucous membrane.

(6) Frog's eggs or sago-like clumps which have often been described as characteristic, but which have been shown by Notknagel to be present in other diseases.

The TENESMUS of dysentery is its most characteristic subjective symptom. It is so great that the desire for stool is practically constant and does not seem to be mitigated in the slightest degree by the evacuations. Very often indeed the tenesmus extends to the bladder, giving rise to an almost constant *ardor urinæ*.

The more or less constant straining at stool gives rise to local changes and the anus becomes congested and fissured. It is then spasmodically contracted. Later, as the disease progresses to a fatal issue, relaxation occurs and the orifice stands wide open, the sphincter being paralyzed. Stools then escape involuntarily, and local pain is slight. This condition must not be misinterpreted to mean improvement.

Abdominal pain, usually described as tormina, is present in all cases, and is due to the constant peristaltic movements of the large intestines, especially the transverse colon. It is often colicky and is especially worse after and before stool. In severe cases there is tenderness along the entire line of the transverse colon.

The urine is scanty and contains an excess of solids.

Other symptoms referred to the gastro-intestinal tract are often present, but are not characteristic. The tongue may be furred and moist, or glazed, or covered with a grayish or yellowish coating; or assume a typhoid appearance. Nausea and vomiting are sometimes present, but are rarely sufficient to cause frequent rejection of food. The vomiting is usually limited to the early stages of the disease. When severe and prolonged it is a serious symptom.

The abdomen rarely presents any outward evidence of disease. It may be distended and is frequently tender.

In the diphtheritic variety symptoms of a typhoid character appear. The sensorium becomes clouded, or delirium is developed. The pulse is rapid and weak. The patient becomes rapidly prostrated and dies in collapse.

**Complications and Sequelæ.**—The ordinary catarrhal cases are free from complications and sequelæ. Abscess of the liver is one of the most common complications of the amœbic or epidemic variety. In some cases extension of the inflammation and ulceration to the peritoneum and the perirectal connective tissue leads to peritonitis (circumscribed or diffused), abscess or anal fistula. The cicatrization of deep ulcers may result in the formation of strictures of the bowel. Not infrequently dysentery leaves its victim very susceptible to agencies causing diarrhœa. Pyæmia or septicæmia of different degrees of severity may sometimes be produced, and to these causes the joint troubles which sometimes accompany or follow dysentery have been ascribed. Some have claimed that



these arthropathies are rheumatic in origin; and still others that they partake of the nature of gonorrhœal rheumatism. The occasional secondary occurrence of heart disease has given some support to the former view. Whatever be their nature, they prove a very serious complication, as the inflammation may produce ankylosis. Paralysis sometimes occurs after dysentery. It is usually limited to the lower extremities, and was at one time regarded as of spinal origin. It is now generally accepted that it is due to an ascending neuritis.

**Diagnosis.**—Dysentery has such a well-defined symptomatology that its recognition would appear an easy matter. And yet errors frequently arise, apparently from sheer carelessness and indifference. The most frequent error is that of calling an ordinary diarrhœa with more than the usual amount of straining at stool, dysentery. To bear this error in mind is to guard against it.

*Typhoid fever* may simulate the diphtheritic type; but this disease shows brain symptoms and a typical temperature curve, and is accompanied or preceded by epistaxis and bronchitis. Dysentery is characterized by a lower grade of fever, absence of the splenic enlargement and the characteristic roseolous rash of typhoid fever.

*Local conditions about the rectum* may not infrequently excite loose and bloody stools with considerable tenesmus. In such cases there are always data which at once throw doubt on a diagnosis of dysentery and suggest a physical examination, which in turn reveals the true nature of the disease.

**Prognosis.**—The prognosis of the ordinary catarrhal dysentery is favorable. The epidemic types are always dangerous. The mortality may run as high as 60 or 70 per cent., and as low as 10 per cent. The appearance of fecal matter in the stools is generally of good omen; but if scybalæ only appear without the subsidence of other symptoms, but little comfort can be derived from the fact, for the fecal masses may have been retained for some time, and are now discharged because of the relaxation of the sphincter. Complicating peritonitis is, of course, dangerous, if not absolutely fatal.

Mild cases of the disease may run their course in about eight days to three weeks. The severer types may pursue a course of six or eight weeks before recovery is finally established.

A less favorable prognosis must be given in the extremes of age.

**Treatment.**—During an epidemic of dysentery every available means should be employed to prevent the spread of the disease. Personal and local cleanliness is of great importance. All stools should be promptly and thoroughly disinfected before being disposed of. The digestive organs should be preserved in the best condition, and all water or milk, or other fluids taken as drink, should be boiled. Fruits and vegetables, unless in perfect condition, should be avoided, or better all



should be cooked before eaten. Rest in bed is the first item of importance in the care of a patient suffering from dysentery. The use of a bed-pan or of absorbent cotton to receive the discharges should be insisted upon, as frequent rising to stool is a great disadvantage to the patient. A cotton-wool pad upon the abdomen secured by a firmly applied broad binder often proves a great comfort to the patient. If tormina is severe, frequently renewed hot fomentations assist in the control of the pain, and early in the attack may favorably influence the inflammatory process, if persistently applied. The *food* should be exclusively liquid; milk in the many forms in which it may be prepared is to be recommended. Farinaceous gruels and animal broths may sometimes be added to the dietary with advantage. Rice and barley-water and whey are nutritious and answer well for drink. A return to solid food must be made with caution. In protracted or chronic dysentery a proper diet is indispensable to improvement. *Stimulants* are of advantage if prostration is great, the stronger alcoholics, such as whiskey or brandy, being preferable. Whether it is ever wise to adopt the common practice of clearing out the intestines in the early stage of the disease by means of a saline purge, is a question upon which diverse opinions will be expressed. It is advisable to reserve this plan for exceptional cases. Intestinal irrigation, if properly performed, is a most valuable adjunct. Solutions of nitrate of silver, alum, salicylic acid, carbolic acid, and mercuric chloride have been especially recommended for this purpose. Whatever benefit is secured from these irrigations seems to be due rather to the free cleansing of the large bowel than to any specific influence of the agents employed upon the mucous membrane of the intestine. Very good results follow the use of simple boiled water, listerine diluted with four or five times its volume of water, or a solution of boracic acid in water, ten grains to the ounce. The method of administering injections is important. The patient should be placed on the side without a pillow and with the hips well elevated, the fluid should be allowed to flow very slowly, with a temporary arrest as soon as an inclination to its rejection is developed; when this subsides the injection should be gradually resumed. It is important to introduce as large a quantity as the intestine will contain without exciting pain. This plan is entirely safe except in the latter stages of the disease after deep ulcers may have formed. The irrigation should be repeated every six or twelve hours. Inability to retain the fluid is sometimes overcome by the previous injection of a little of a weak cocaine solution. Osler reports beneficial results from irrigation with solutions of the sulphate of quinine, 1-1000 to 1-5000, in amœbic dysentery; he states that the amœba are rapidly destroyed. It is proper to say that valuable as is this local treatment it is seldom required by the cases seen in this vicinity, providing they have had proper treatment from the beginning of the attack.

Among remedies for dysentery *mercurius corrosivus* easily retains the first place. It has long been employed in homœopathic practice and has of late become popular with the old school, largely through the writings of Ringer, Phillips, and others. The bloody mucous stools, the tormina and tenesmus (which are the prominent clinical phenomena) are highly characteristic of *mercurius corrosivus* provings. Clinical experience has demonstrated its usefulness in the most serious forms of the disease; *mercurius dulcis* being preferred for the milder cases, *i. e.*, cases with the minimum amount of pain, tenesmus, etc. In the early stage of the disease, often before the condition is recognized as dysentery, *aconite* and *ferrum phosphoricum* are useful. The former medicine is especially called for by the *aconite* causal indications, and the prominence of the fever; *ferrum phosphoricum* is suited to very similar conditions in which fever is subordinate. This medicine is excluded if tenesmus is a marked symptom. It seems especially valuable in the dysenteric attacks of children.

*Cantharis* has not received proper credit as a remedy for dysentery. It is well suited to the worst cases, especially the epidemic form. The discharges from the bowels are bloody, slimy and mixed with flakes resembling scrapings of the intestines. There is tenesmus of both the rectal and vesical sphincters and abdominal pains of a colicky character. These pains may be intensely burning and lancinating. Five drops of a good tincture in four ounces of water may be administered in teaspoonful doses every one-half to two hours as required.

*Arsenic* is called for in the later stages of severe cases of epidemic dysentery by its well-known indications. *Colchicum* should be studied in connection with this remedy. *Arnica*, *arsenic*, *baptisia*, *rhus toxicodendron* are useful for cases developing typhoid symptoms. *Ipecac*, *belladonna*, *colocynth*, and *aloes*, are useful medicines for the milder attacks of sporadic dysentery. *Nux vomica* preceded or accompanied by *aconite* is, with proper general care, often sufficient for their relief. Concerning the value of *colocynth* in dysentery there has been considerable difference of opinion. Many hold that notwithstanding its symptomatic correspondence to some cases, it rarely if ever suits the disease, the symptoms being more prominently neurotic than inflammatory in character. The prominent indication for this medicine is abdominal pain of a cramping colicky character, relieved by pressure or bending double. It is the writer's experience that pains of this character are often present in dysentery, often relieved by *colocynth*. The *arsenite* of *copper* is valuable for the same symptom. *Aloes* is strongly suggested by tenesmus, which it sometimes relieves even after the failure of *mercurius corrosivus*; the *aloes* patient has a great deal of griping pains before the stools, which contain much jelly-like mucus.

*Opium* in some form is in general use for the control of the pain and

is often a great comfort to the patient. It is an error, however, to use it to "check the bowels." It is seldom necessary in cases treated judiciously from the beginning. Morphia in doses of one-eighth to one-quarter of a grain is the best form of administration.

During convalescence attention should be bestowed upon the diet, the amount of exercise taken and especially upon the condition of the bowels. These must be restored to a normal condition before the patient passes from observation, otherwise permanent weakness may result.

## MALARIAL FEVERS.

**Definition.**—The Italian word malaria, at one time used to signify bad air in the broadest acceptation of that term, is now restricted to that specific infection manifesting itself in various ways, but which is the cause especially of certain well-defined types of fever attended by characteristic blood changes and alterations in the adenoid tissues. The origin and nature of this poison are questions which have attracted much attention, and which now seem to have been solved. The various theories advanced as to the nature of the poison have given way to the prevailing germ theory which has been accepted by a majority of those best qualified to judge.

**Etiology.**—The conditions favorable to the development of malaria are first, certain telluric states. It is a well-established fact that the vast majority of cases of malarial infection occur in poorly drained, low-lying, moist regions, where vegetable growth is abundant. Other conditions are essential, however, as shown by the occasional absence of infective power in this pestilential appearing marsh, and the disappearance of the poison from marshes once known to be breeding grounds. The conditions of soil and atmosphere necessary to the production of the malarial element appear to be: (1) Decomposing vegetable matter; (2) the presence of moisture, which need not be in large amount; (3) a proper temperature, *i.e.*, an average temperature for the twenty-four hours of at least 60° F. The higher the temperature, however, the more favorable is the prospect of the generation of the specific element. The tropical regions present in the highest degree a proper combination of these essential conditions. The malarial diseases, however, are widespread, although gradually becoming more restricted as to the area of their development. Along our Atlantic seacoast the number of foci of development is gradually growing less. New England, once so much afflicted by it, is gradually being emancipated. This city and the region bordering on the two rivers—the Delaware and the Schuylkill—at one time highly malarial districts, are now comparatively free. Even in the old-time malarious regions of our sister states, east and south, malarious manifestations are less common. Further south, in Mississippi, Louisiana, Arkansas, Texas and Florida; also in Mexico and Central America, malaria is endemic and at times assumes a pernicious and perhaps epidemic character. The disease is almost unknown in certain regions of our West and Northwest. Abroad, the hotbeds for the propagation of the malarial poison are Russia (in its southern warmer portion) and Italy. In the latter country



the Roman Campagna stands as an example of a typically malarious region. Malarial affections are not common in Great Britain, Germany or France. Accidental foci of malaria are occasionally developed by the turning up of earth, the opening of a sewer, the draining of a swampy land, by a period of dry heat after heavy rains or when cultivated regions are abandoned. Impure bilge-water has been known to develop the disease in vessels. Malaria is often endemic where salt and fresh waters commingle, therefore along certain seashores, notably on the borders of the North Sea, along the coast of Africa, etc. The manner of introduction of the poison into the body is not certainly known, but it seems probable that it is mainly through the respiratory apparatus. Perhaps the drinking water may contain it.

That the poison possesses weight is shown by the greater immunity of those living in the upper portions of dwellings. It may, however, be driven by currents of air up the sides of mountains to a considerable height. The inhabitants of certain hamlets have been known to be attacked in numbers when the wind blew from the direction of a neighboring marsh. The growth of a grove of trees or the building of a wall between a marsh and a town has been known to greatly diminish the disease.

Those desiring to avoid contracting malaria should remember that the poison rises from the ground in the moisture of the night, and is dissipated by the sun in the morning. They should therefore retire to their dwellings early and sleep in the upper rooms, remaining indoors until the morning sun has accomplished its mission. All excesses must be avoided. The drinking water should be boiled.

All ages and both sexes are attacked. One attack predisposes to another, the manifestations perhaps continuing at intervals for a lifetime if the individual is unable to leave the infected district. Efforts to demonstrate malaria to be of bacterial origin have thus far failed. In 1879 Klebs and his Italian colleague, Tommasi Crudelli, isolated a bacillus, the so-called *bacillus malarie*, which they supposed to be the causative agent, but their observations have not secured corroboration. More recently (1880) Laveran, at that time a surgeon in the French army, described an organism found within the red blood corpuscles and in various free forms in the blood. That found in the blood cells has been named the plasmodium. The facts as stated by him and confirmed by Celli, Marchiafava and Golgi abroad and by Osler, Dock, James, and Councilman in America, are as follows:

**THE SPECIFIC ORGANISM.** The micro-organism of Laveran belongs to the protozoa and to the group designated *hæmatozoa*. These parasites of the red blood corpuscles are frequently met in the blood of birds and some of the lower forms of animal life, especially the frog, fish, turtle, etc. As met in the blood of persons suffering from malarial fever

this parasite presents varieties in size and form. This fact has led to the belief upon the part of some observers that special species exist, each one being possibly related to a single form of fever. Marchiafava and Bignami assert that both the biological and morphological peculiarities of the parasites differ so much in the various types and species of fever, that the existence of several varieties seems probable. Laveran opposes this idea, advocating the doctrine of unity and a belief in the polymorphic character of the organism. He considers the various forms of malarial fever as the result of action of the several forms of the parasite, and partly perhaps of the condition of the patient. According to Laveran the blood of persons affected by malarial fever contains one or more of the following forms: (1) spherical amœboid bodies containing nuclei; (2) a crescentic form with nuclei; (3) rosettes; (4) flagellated bodies.

The method of examination advised by the discoverer is as follows: Receive a drop of blood upon a cover-glass, dry over a flame, stain with a saturated solution of methyl-blue, or, if preferred, with gentian-violet. The organisms will be found in the red blood corpuscles or in the blood plasma. The flagella are exceedingly delicate and cannot be detected if the specimen is not fresh. Their motion assists in their detection, which is also easier if oblique illumination is used. The number of ciliæ visible varies from three to eight or nine. Councilman found a larger number of this form in blood drawn from the spleen. The other forms can be examined in specimens which have been preserved for some time. In a recent study of the subject by Marchiafava and Bignami they state that the hæmatozoa appear as amœbæ which develop in the interior of, and at the expense of, the red corpuscles, converting the hæmoglobin into melanin, or quickly developing a toxic necrosis, with a resulting anæmia. According to these observers the amœbæ complete within the corpuscles a cycle of existence represented by (1) a motile unpigmented form; (2) becoming pigmented as development proceeds; (3) segmentation forms, representing the multiplication of the organism. With the liberation of the spores resulting from the segmentation, they are replaced by fresh amœbæ which in turn invade other blood corpuscles. There is a parallélism existing between the recurrence of the paroxysms and the completion of the life-cycle of the parasite, the beginning of the paroxysm being coincident with the segmentation. While it has not yet been demonstrated, it seems probable that during the period of development of the amœbæ, toxic substances are formed; during reproduction, a pyrogenic toxin; during intra-corpuscular development, in fevers of high grade, they elaborate substances which possess the property of causing necrosis of the red cells, separating the hæmaglobin from the protoplasm, and exciting alterations in internal organs. The pathogenic qualities of these organisms also warrant the assumption of various types or species

of fever. The following varieties are recognized as established (Golgi, Marchiafava, Bignami): The amœba of quotidian fever; the amœba of tertian fever; the amœba of summer-autumn tertian fever; the amœba of quartan fever. The pernicious forms of fever are due only to the amœba of quotidian and summer-autumn tertian fever. If these observations prove correct the practical outcome is important, *i.e.*, it becomes possible to diagnosticate at once cases which may prove pernicious from those which never do.

Marchiafava asserts that the malarial organism is subject to seasonable variations, at least in Italy. In summer the red corpuscles are occupied, amœboid bodies being associated if symptoms of perniciousness are present, crescentic and filiform shapes being more common in autumn. Inoculation experiments have been followed by success in rabbits, paroxysms of intermittent fever being developed, followed by pigmentation of the spleen.

**Morbid Anatomy.**—The lesions incident to malarial poisoning involve especially the blood, the spleen and the liver. In the very acute forms of malarial infection the blood contains masses of blackish or brownish pigment matter, either free in the blood plasma or within cell-bodies resembling leucocytes, or in the vascular endothelium. This pigment matter accumulates in the spleen and liver, where it is found abundantly upon post-mortem examination, giving to the organs a characteristic brownish or blackish color. Such discoloration is associated with enlargement of these organs and chronic interstitial changes. The spleen may rupture spontaneously or as the result of traumatism. Pigment masses may also obstruct the finer bloodvessels. In the severer types hæmorrhage into or from the mucous membranes may take place. The morbid changes in malarial infection are due to the disintegrating action of the parasite upon the red blood corpuscles, and probably also to the action of toxic agents elaborated in the course of its life-cycle.

Anæmia is a constant feature of malarial infection and is the result of the destruction of red blood cells by the hæmatozoa.

### INTERMITTENT FEVER.

Intermittent fever is a widely distributed affection, and the most frequent form of malarial infection. It is characterized by repeated periodical paroxysms, consisting in the typical form of three stages, *i. e.*, chill, fever and sweat. The frequency of repetition of the paroxysms has led to the application of certain terms. A quotidian fever is one in which a paroxysm recurs every day; a tertian fever is one which occurs every other day; and a quartan fever occurs every fourth day. These are the more common types. Others are recognized, but they are modifications of those mentioned. A double quotidian is one of the most frequent of these irregular forms. It presents two paroxysms daily, one being



generally more severe than the other. The severe paroxysms usually occur in the forenoon. In the double tertian there is a paroxysm daily, but there are two distinct series of paroxysms which may be differentiated, those which resemble each other occurring at forty-eight-hour intervals. The dissimilarity in the two series relates especially to their intensity, or absence of one or more stages of the paroxysm. A repetition of the paroxysms at intervals of seven days has been reported, but such a variety must be rare.

Each form presents certain peculiarities as to time of occurrence, etc. The quotidian form is more apt to have a morning paroxysm; the tertian paroxysm occurs late in the forenoon, while the quartan seldom appears until late in the day. It must be remembered that there are many exceptions to these statements.

The incubation period is very variable. In intensely malarial regions a few hours of exposure is frequently followed by a paroxysm. Ordinarily, a week or more is required. Cases in which the disease has been said to develop long after exposure are open to suspicion.

**THE PAROXYSM.** The approach of the chill is often heralded by headache, backache, yawning, weakness and perhaps nausea and vomiting. The use of the thermometer at this time will frequently result in finding a slight rise of temperature. The chilly sensations are apt to begin in the back and gradually extend to the entire body. The patient shivers; the teeth chatter; the voluntary efforts are attended by trembling, with finally shaking of the entire body. The features are pinched and with the finger ends may look bluish. The voice is husky and the breathing quick. The skin becomes roughened (goose-skin) and cool, and upon the application of the surface thermometer is found to be much below normal in temperature. These symptoms, it is apparent, are all due to spasm of the superficial vessels developing a high degree of bloodlessness of the superficies of the body. At the same time the internal temperature has been constantly rising, and at the height of the chill the rectal temperature frequently attains 105°–106° F., or even a higher point.

**ATTENDING SYMPTOMS.** Severe pain in the head, back and limbs, nausea, vomiting, thirst, increased clear urine frequently voided, and a rapid pulse of high tension attend. The mind is seldom disturbed. The chill may last from a few minutes to two or three hours, according to the intensity of the case. The chill is seldom well developed in children, and is often in them attended by convulsions; indeed the latter may take the place of the chill. The approach of the hot stage is indicated by flushes of heat. The transition is usually gradual. When it has become fully developed the collapsic symptoms of the period of chill will have fully disappeared, the face is flushed, the skin turgid and hot, the patient is restless, the pulse full and bounding, and the temper-



ature, if changed at all, is higher. Not infrequently the highest temperature is reached at the termination of the cold stage.

Nausea and vomiting are even more frequent and troublesome than in the cold stage. Thirst is very great. The urine is scanty and highly colored, and, in the pernicious forms of fever, may even be suppressed. The spleen is appreciably swollen. The hot stage continues from a half hour to from four to six hours, and occasionally longer. The longest duration is in the quotidian, and the shortest in the quartan form. The approaching termination of the hot stage is indicated by the appearance of a moisture upon the forehead and its gradual extension to the entire body. Like the preceding stages this one varies greatly in intensity, from a slight moisture to a drenching sweat lasting from a half hour to one or two hours. With the final stage there is relief of all symptoms. The fever symptoms quickly abate, the pains disappear, and the patient is apt to fall into a refreshing sleep. When he awakes, he feels very weak, though otherwise well.

THE INTERPAROXYSMAL PERIOD. Unless many symptoms occur the patient does not often complain of ill health between his paroxysms. A careful study of the individual at this time will, however, often furnish symptoms mainly subjective, which will prove of use in the selection of remedies. If intermittent fever is long continued or frequently repeated, the patient becomes anæmic, the skin appears jaundiced, and the tissues pigmented; the spleen and perhaps the liver become enlarged. Indeed the various evidences of chronic malarial infection are developed.

In this latitude the quotidian and quartan types occur more frequently than any of the others. Occasionally the paroxysms occur with each return a little earlier than the one preceding. They are then spoken of as anticipating; if a little later each time, they are called postponing. Frequent recurrence in the same individual usually results in irregularity of course, their regularity affecting time of occurrence, intensity and the order of succession of the several stages of the paroxysm, etc.

IN CHILDREN. The symptomatology of the disease as seen in children warrants notice. The pernicious form is not uncommon. The chill is slight or absent, and may, as already stated, be replaced or attended by convulsions and coma. The hot stage is marked and prolonged. If convulsions have occurred in the cold stage, coma may follow in the hot. If convulsions have not appeared in the cold stage they may now develop. After such serious symptoms have continued for some hours, and perhaps the nature of the affection is not recognized, the child improves and the intermission is established. Such attacks are fatal as a rule, especially if repeated. In children the intermissions are rarely perfect, gastric or intestinal symptoms continuing and the intermittent lapsing into a remittent form of fever. In many of these cases a search

for Laveran's organism in the blood is the only means to a proper diagnosis.

**Diagnosis.**—This is generally made without difficulty. The intermittent form of fever occurring in septic infection, and in connection with some cases of pulmonary phthisis, is frequently mistaken for a malarial fever. Careful physical exploration is sometimes necessary in order to make clear the nature of the fever. In doubtful cases the practitioner should search for Laveran's organism, which test is of positive value.

**Prognosis.**—The prognosis is good in simple uncomplicated cases of intermittent fever. The possibility of transformation into the pernicious type, or development of the malarial cachexia from long continuation of the disease, must be considered.

### REMITTENT FEVER.

**Nomenclature.**—This form of malarial infection is recognized in different portions of the world by a great variety of names, such as bilious remittent fever, bilious continued malarial fever, gastric African, Southern and Western fevers, etc.

**Definition.**—A continued form of malarial fever marked by remissions. Its duration may vary from a few days to a few weeks. The fever may sometimes be attended by intermissions either in its beginning or termination.

**Etiology.**—The cause of this fever is malarial infection. All forms of malarial manifestations are probably due to the same cause, which appears to be modified in its character and operations by a variety of conditions, of which the most important appears to be temperature. Remittent fever is especially prevalent in regions with a high average temperature, consequently prevailing in its most intense form in the tropics.

The prevalence of this fever in regions where intermittents are endemic, the frequent conversion of an intermittent into a remittent form, and *vice versa*; the similarity in pathological lesions, all suggest the relationship of these two forms of fever to a common cause. The distribution of the disease is extensive, occupying the entire equatorial portion of the globe, and shading out into the temperate zones. Remitting fever of non-malarial origin occurs occasionally, especially in children, and is often attributed to malarial infection. There is usually little difficulty in distinguishing cases of this character.

**Morbid Anatomy.**—The tissue changes of remittent fever are much the same as those described as occurring in intermittent fever, but of a higher grade. This difference in degree is conspicuous in the alterations found in the blood. The greater destruction of the corpuscular elements in remittent fever, and the presence of pigment granules in the blood and various tissues, is the most conspicuous change. The pigmentation is well marked in the grave and protracted forms of the disease.

The spleen is always swollen (due to hyperæmia), and if the attack is much protracted, or there are repeated attacks, the organ remains permanently enlarged because of hyperplasia of its elements. In the great enlargement attending severe attacks, the organ is often literally stuffed with pigment. The liver is swollen and hyperæmic, and of a slate or bronze color. A slate color of this organ is considered as quite characteristic of malarial diseases.

The gastro-intestinal mucous membrane is frequently the seat of a marked hyperæmia or actual inflammation. The tissue is thickened or softened, and the glandular elements swollen and pigmented. The solitary glands of the colon, according to Hertz, are sometimes inflamed, the seat of partial suppuration and the changes peculiar to dysentery are at times extensively developed. Hertz also states that in cases of the adynamic form, that there is less hyperplasia of the various organs noticeable, and a more marked degeneration of the muscular system, especially of the cardiac muscle, which is soft and easily torn.

**Clinical Course.**—The onset is usually abrupt, being preceded in some instances for a day or two by oppression at the epigastrium, anorexia, a little nausea, general pain and bad feeling. The initiatory chill is not generally very severe, not as severe as is usually met with in intermittent fever. The sense of coldness is very generally distributed over the entire body at once. It is followed by a rise of temperature to 101° to 103° F., or higher in some instances. The fever continues without any decided remission for from twelve to forty-eight hours, and is usually attended by vomiting, which may have begun during the chill. The patient is restless, flushed, complains of headache, a total loss of appetite, and especially of pains at the epigastrium. The temperature falls to about 100° F., and if the attack has been a slight one, the patient may feel like getting up. The remission is indicated by a relief of the more distressing symptoms. The fever does not, however, disappear. Although the relief is considerable it is never so great as that obtained in the intermittent fever. He still has no appetite, and there is a variety of little symptoms remaining, and these annoy him. The remissions occur at variable intervals, but nearly always in the morning. Sometimes they may take place twice in the twenty-four hours, oftener once daily. Occasionally the patient may present depressed temperature only at intervals of several days. With the exacerbation, which may be ushered in by a chill, there is an aggravation of the principal symptoms detailed—*i. e.*, vomiting, pain, restlessness, etc. During these periods the temperature often rises to 105° or 106° F. Slight delirium is not uncommon, and a hæmatogenous jaundice sometimes develops in the highest grades.

The duration and severity vary within wide limits. Mild cases recover in from six to eight days, and may, because of their mildness,



be looked upon as of a doubtful nature. If protracted, the daily remissions become gradually less distinct; in fact, the fever is transformed into one of a continued type, the daily variations in pulse, temperature, etc., being no greater than occurs in typhoid fever. With the change of type appears the group of symptoms which resemble the symptomatology of typhoid fever so closely, *i. e.*, delirium, dry brown tongue, sordes, lessening of the vomiting, but a continuation of the epigastric distress. Diarrhœa replaces the earlier constipation, and is attended by distension of the abdomen. The urine is scanty, dark and acid, and it may even be albuminous. The pulse is small and frequent. Prostration may be so great that the patient cannot maintain himself on the pillow, but slides to the foot of the bed. Delirium is present, and is accompanied by subsultus tendinum and picking at the bedclothes. In this stage it is not remarkable that the disease should be mistaken for typhoid fever. If the issue is to be a fatal one, these symptoms continue, attended by a gradually increasing prostration, the patient finally succumbing to progressive asthenia or to some complication. A favorable termination is indicated by the reappearance of remissions and a gradual subsidence of all the symptoms. The temperature is, perhaps, the most reliable guide to the patient's progress. Exacerbations with a gradually mounting temperature from day to day, a shortening of the periods of remission, and a daily occurrence of the period of exacerbation at an earlier hour, and especially the development of the typhoid group of symptoms, all indicate an unfavorable course. The presence of an unusual degree of gastro-intestinal catarrh occasionally leads to jaundice and bilious vomiting. This has led to the recognition of a special form of fever called "bilious remittent," which is supposed to be specific form of fever not related immediately to malaria. The distinction cannot be sustained. The same may be said of the so-called "infantile remittent" fever, which depends upon many causes, such as gastric or gastro-intestinal catarrh, irritation from worms, etc., but more especially upon the action of the typhoid poison which develops a typhoid fever of rather irregular type, and which with sufficient care can generally be easily diagnosed.

**The Diagnosis** of remittent fever is sometimes beset with difficulties; but these have been lessened by the invaluable researches of Laveran and other observers who have followed in his steps. We now have much valuable evidence respecting the diagnostic value of the organisms as described by him, evidence coming from all portions of the globe. Dock, Osler and others in this country have furnished us with valuable information, and Carter has written an interesting monograph in which he speaks of the value of blood examination in the fevers of India. We believe with Osler that there are probably but two forms of continued



fever existent, "the one due to the typhoid, and the other to the malarial infection." The especial difficulty in diagnosis arises when the malarial fever becomes continuous and typhoid symptoms develop. The organism of Laveran, the free pigment in the blood, the usually abrupt origin of remittent fever, the marked remissions, the hepatogenous character of the jaundice, which can frequently be determined, and the infrequency of hæmorrhages in remittent malaria, are some of the conditions aiding to a correct differentiation.

**Prognosis.**—The prognosis is good especially in this latitude, where the disease is of a mild type. It is less favorable in highly malarious regions in the South. A certain percentage of remittent fever cases assume the pernicious type and the prognosis is then correspondingly grave. The previous condition of the patient, especially as to lesions of vital organs (heart and kidneys especially), is of importance in determining upon a prognosis. Inflammatory complications of quite a variety have been observed, and according to their location involve the prognosis.

### PERNICIOUS MALARIAL FEVER.

The pernicious variety may present itself in any of the recognized type of malarial infection. It is most frequently met with in the quotidian and tertian varieties of intermittent fever. The pernicious character may not be apparent until the disease has been thoroughly established, or it may develop in its worst form at once.

We recognize several varieties. The following are the most important: (1) Delirious; (2) comatose; (3) gastro-enteric; (4) algid; (5) hæmorrhagic.

(1) **THE DELIRIOUS FORM** is characterized by an unusual development of delirium during the febrile stage of an intermittent, or the period of exacerbation of a remittent fever. It is apt to be associated with or preceded by headache, vertigo, ringing in the ears, flashes of light, and other cerebral symptoms. The delirium is of a most active character, the patient having a strong tendency to violence. There may be viciousness or hilariousness; the patient singing, yelling, or crying. The circulation is excited, the pulse being bounding, the face flushed, and the eyes injected. The temperature is usually very high. In place of the delirium, epileptiform or tonic spasms may occur after several attacks. The patient dies in coma or collapse.

(2) **THE COMATOSE FORM** is marked by the development of serious cerebral symptoms, the most important of which is a more or less rapidly developing coma coming on during the period of aggravation of a remittent or during the hot stage of an intermittent fever. These symptoms may be a feature of the first paroxysms or any subsequent one. The unconsciousness may be partial or complete, and continue as long as

eighteen to twenty-four hours, terminating in death or recovery. Improvement is often shown by the appearance of sweating. If the patient recovers from the first attack, a second or third one, if it occurs, is more likely to prove fatal. During the coma the breathing is stertorous, the pulse slow, the face flushed, the pupils dilated, and the temperature high ( $105^{\circ}$  to  $106^{\circ}$  F. or more). The urine may be retained.

If a paroxysm has preceded the one in which the coma has developed, it is probable that symptoms of cerebral irritation have been present. In a region in which pernicious varieties of malarial fever exist, the occurrence of vertigo, headache, thickness of speech, with inability to think and speak connectedly, should excite suspicion as to the character which the next paroxysm will develop, and energetic treatment must be instituted. It is in this variety that the interesting condition of apparent death sometimes develops. Some patients under the circumstances are unconscious, others are cognizant of all that is transpiring about them, but unable to utter a sound or move, even in response to powerful stimuli. The coma has been known to continue for days prior to death.

(3) GASTRO-ENTERIC FORM. At the same period as in the preceding varieties, the patient may be attacked not by delirium or coma, but by violent vomiting and purging. The characteristic of the vomit and stools is the presence of blood in varying amount. Both the vomited matters and the stools are apt to be thin; even rice-water discharges are not rare. These thin stools tinged with blood resemble beef-juice brine. Occasionally the stools consist of almost pure blood. In certain epidemics, the symptoms are wonderfully like those of Asiatic cholera. This variety is often rapidly fatal from collapse, the entire duration of the illness being but a few hours. In considering their symptoms and course, one can hardly go astray after studying the phenomena of cholera Asiatica.

(4) THE ALGID VARIETY is individualized by the low temperature, cool surface even when the patient complains of burning heat, and the gradually progressing failure of the circulation. Such an attack may be initiated by gastric irritation, but the symptoms of circulatory failure become and remain the prominent feature. Algid paroxysms are highly fatal, seldom being arrested after having been fairly developed.

(5) HÆMORRHAGIC FORM. Hæmorrhage seldom occurs in malarial fever unless the attack is protracted. If so, bleedings may occur from the mucous surfaces. Then hæmorrhages are especially apt to occur during the sweating stage, and may be from any of the mucous surfaces. Rarely the sweating stage is so copious or protracted as to lead to dangerous or fatal results. There is attending prostration, labored respiration and failure of the circulatory system. The patient may be lost if these "colliquative" sweats are repeated once or twice.

## MASKED INTERMITTENT FEVER.

This term has been applied by a number of authors to certain febrile periodical manifestations, especially certain neuralgiæ due to malarial infection. The supraorbital branch of the fifth nerve is most frequently attacked, and this form of neuralgia is sometimes spoken of as "brow-ague." The sciatic, crural, or brachial nerves may be the seat of intermittent pains. Visceral neuralgias due to the same cause have been reported. Instead of neuralgia, intermittent convulsions, headaches, hæmorrhages, paralysis of motor or sensory nerves, cedema, and various gastro-intestinal symptoms have been attributed to malaria. The evidence of a causative relationship has not been proved in all instances. There is too much of a tendency in malarious regions to ascribe many conditions, which are not clear etiologically, to the common enemy—malaria.

## THE MALARIAL CACHEXIA.

The malarial cachexia is a condition resulting usually from the long-continued action of the malarial poison, although at times all of its features are developed after comparatively short attacks of intermittent or remittent fever. It is sometimes observed in its most characteristic manifestations in persons who have at no time had febrile paroxysms. This occurs, however, only in intensely malarial regions. Profound anæmia and enlargement of the spleen are its most prominent characteristics. The symptoms are largely due to anæmia, which is such a marked element in all cases of malarial disease. There is cardiac palpitation, shortness of breath, rapid feeble pulse, and anæmic murmurs in the blood-vessels. The temperature is variable. Hæmorrhages may occur from any of the mucous surfaces, or into the retina, lung tissues, etc. Fatal hæmatemesis is not rare when the spleen is much enlarged. The splenic development is often quite as great as in leucocythæmia. The liver is also enlarged and both are pigmented.

**Treatment of Malarial Infection.**—INTERMITTING FORM. The patient suffering from intermitting fever receives as a rule too much of specific medication, too little of care and attention to the general health. If still in the region in which the infection has been contracted, the patient should be put at rest in one of the upper rooms of the house until the paroxysms have been checked, and should then for some time retire to his upper room before sundown and remain until the sun is well up in the morning. By this method increased dosage with the poison may be avoided. While it is not necessary in ordinary uncomplicated intermittents to keep the patient in bed, a marked degree of rest is advantageous. The diet should be nourishing, easily digestible and largely liquid. The surface of the body should be well sponged daily and general personal and local cleanliness enforced. It is a wise precaution to boil all water taken and use only well-cooked food.



**MEDICINAL TREATMENT.** The value of quinine when properly administered in the treatment of intermittent fever is so much greater than that of any other known remedy that it is simply inexplicable that any clinician should hesitate to use it. Few would hesitate were it not for the reason that substantial doses are often required. The question of dose is so inextricably associated with the homœopathicity of a prescription in the minds of many practitioners, that any remedy which must be given in a large dose is to their minds unhomœopathic and unadvisable. This position is so manifestly absurd to the minds of rational physicians that it seems hardly worth contending against. That quinine is homœopathic to typical intermittent fever is undoubted, and that in some doses it is capable of controlling nearly all cases of this character without developing unpleasant symptoms or leaving sequelæ, is equally true. While not believing that quinine is the best remedy for all cases of intermittent fever, it should certainly be our first choice for the majority. In doses of from fifteen to thirty grains given during the four hours preceding the chill, it is an efficient antiperiodic, promptly controlling recent cases. This influence of quinine is so certain that many consider a case which cannot be arrested by this drug as not malarial in character. Hahnemann recommended the use of bark alone in recent cases of intermittent fever. Those who have had the most experience in this disease have arrived at the same conclusion respecting its value. As to the dose there are many opinions. Osler (allopathic) says: "I have a number of charts showing that grain doses three times daily will prevent the paroxysm, but not always with the certainty of the larger doses." The plan followed in recent years by the author is upon the patient's first visit, if another medicine is not clearly indicated, to prescribe two grains of the first decimal trituration of *bisulphate of quinine* every two hours until the time of the next expected paroxysm. If the paroxysm does not appear, the medicine is to be continued at longer intervals for several days. If there is no improvement, a full antiperiodic dose is prescribed previous to the next paroxysm, and this dose should seldom be less than eight or more than fifteen grains. If the paroxysm is anticipated, the medicine should be continued for a time in decreasing doses, but administered in the same manner, *i.e.*, practically in one dose three or four hours before the paroxysm. Even if quinine is given in large doses, much relief may be secured from certain symptoms by the use of symptomatically prescribed medicines.

*Ipecac.* often relieves the nausea and vomiting, and is also a remedy of value against recent agues.

*Belladonna* often diminishes the headache of the hot stage, and is especially valuable in the disease as it appears in children. If the chill is severe and the succeeding symptoms suggest the pernicious variety, such medicines as *arsenicum*, *veratrum album* and *camphor* may be indicated, and supplement the action of quinine.



Quinine acts especially and undoubtedly as an antiparasitic, arresting the growth or destroying the products of the plasmodium malariae. The success attending this poisoning of the specific germ of malaria is greater than the success attending the germicidal treatment of other infectious diseases. This seems explainable by the presence of the germ, in its active varieties, in the circulating fluid only, therefore more accessible to the action of drugs. Binz (*Centralbl. f. d. Med. Wiss.*, 1867, p. 305), has shown that a solution of quinine, one to twenty thousand, has a killing influence on certain infusoria. A strength of one in seven or eight thousand can be secured in the blood by the administration of ten grains of quinine.

TREATMENT OF MALARIAL FEVER OF REMITTENT TYPE. In the remittent form of malarial fever the patient should remain constantly in bed and upon a fever diet, great care being exercised to avoid disturbance of the stomach, which organ is often very irritable. All water should be boiled and all food well cooked. To diminish the risk of increased dosage of the poison it is best to select a room for the patient upon the upper floor of the house. For the irritable stomach the patient may receive ice, carbonated water, pieces of ice sucked and swallowed as soon as smooth, or iced champagne. Externally, frequently repeated fomentations, or a mustard plaster to the epigastrium, may afford some relief. If prostration exists in sufficient degree stimulants may be administered. All food and stimulants should be given in definite quantities at regular intervals. Quinine is the essential remedy also in the remitting form of malarial fever, although better results attend general symptomatic prescribing than is the case in the intermitting form.

Large doses of quinine are seldom necessary, the first trituration (given in the form of compressed tablets), in doses of three grains, repeated every one to three hours, usually doing all that quinine is capable of accomplishing. If the remissions are very marked, and the diurnal acme represented by a high temperature, it is well to give enough quinine during the remission to secure a more moderate range later. If quinine does not prove a satisfactory remedy, or if indications for another remedy are present, a selection from among the following may be made: *Aconite* or *veratrum viride*, if during the aggravation there is high arterial excitement, restlessness and anxiety; either of these medicines in the first decimal dilutions prove useful. Occasionally *belladonna* proves a better medicine for this class of symptoms, especially in children when there is drowsiness and a full, rapid pulse. *Gelsemium* is indicated in cases in which the temperature does not run as high; the patient is greatly prostrated from the beginning, has a soft pulse, cool extremities, and may complain of much headache and of double vision. More frequently indicated remedies are *arsenic*, the *arseniate of quinine*,

*bryonia* and *ferrum phosphoricum*. The first two medicines in this group are especially valuable. *Arsenic* is indicated by a high grade of gastric irritability, perhaps diarrhœa, rapidly progressive anæmia and prostration, œdema, nightly aggravation and thirst. The *arsenate of quinine* is best suited to remitting fevers when there is chilliness or a chill at some hour during the day, thus approximating to the intermitting type. It is also valuable if the type changes from the remitting to the intermitting type. *Bryonia* is frequently valuable when there is a good deal of pain in the back and extremities, gastric irritability, which is aggravated by motion; a dry mouth and thirst for large quantities of water. *Ferrum phosphoricum* is most useful for children and when pectoral complications exist.

A typhoid condition may develop in which case a choice of medicines may be made from those recommended for typhoid fever. In the *pernicious* variety the patient is oppressed by a toxic dose, and *quinine* is the only remedy; at least, one should not subject his patient to the risk attending the use of other medicines, as their action is exceedingly uncertain. Large doses (twenty to forty grains) should be administered between the paroxysms, preferably hypodermatically. Pills of this medicine should not be trusted, as they frequently pass the digestive tract undissolved. In addition to quinine, it is wise to prescribe whatever medicine appears best suited to the case. This will vary with the form. In the *algid* variety, *veratrum album* or *camphor*; in the *choleraic*, the same medicines, also *cuprum*, *arsenic* and *arsenite of copper*. For the *hæmorrhagic* variety, *phosphorus* and *crotalus* may be helpful. Stimulants and the most careful feeding are necessary. Everything must be subordinated to the introduction of a sufficient quantity of quinine.

CHRONIC MALARIAL CONDITIONS. It is in the chronic manifestations of malarial poisoning that remedies carefully chosen upon the homœopathic principle produce the best results. How often are we able after long abuse of quinine (for, as in the case of all valuable medicines, it is much used when not indicated) to bring a case to a satisfactory termination by a few well-selected medicines! The range of remedies for this condition is so wide that only a few hints may be given. Of arsenical preparations, *arsenic* is preferred if blood changes are prominent; *arsenate of quinine* for intercurrent attacks of ague; *iodide of arsenic* for the tissue changes, especially weak heart, for the symptoms of incipient phthisis, etc. Anæmia is a prominent indication for each. Of the iron preparations, *ferrum metallicum* is best suited to high grades of anæmia with an irritable vaso-motor system, indicated by frequent flushes occurring in a pale, wretched individual; *ferrum phosphoricum*, if chest symptoms (bronchitis or catarrhal-pneumonic conditions) are present; the *arsenate of iron*, for persistent anæmia, the disease seeming to have spent its entire force upon the blood.

These various medicines have been used most in tablets of the second decimal trituration. If treatment does not give satisfactory results, a change of air is of the utmost importance.

Under all circumstances the nutrition must be carefully considered, exercise regulated, cool spongings employed, and in some cases sprays, douches, etc., are of great benefit in rousing the nutritive activity.

Cod-liver oil or fat in some form is useful. Massage and faradism may also prove a stimulant to torpid nutritive processes, and in troublesome cases, perfect rest in bed for some weeks must be combined with other measures of treatment.

PREVENTIVE MEASURES. Persons who must visit malarial regions, or who reside therein, may avoid infection, for a time, if the region is not highly malarious, by observing the following suggestions: *first*, to remain within doors and, preferably, upon the upper floor of the house from the time the sun begins to lose power in the late afternoon until nine or ten o'clock the next morning; *second*, if the probability of infection seems considerable take ten to fifteen grains of quinine during the twenty-four hours; *third*, eat cautiously, but keep the stomach well filled; *fourth*, avoid undue exercise, exposure to cold, or to the sun's rays, keep the bowels regular, and attend promptly to any symptoms of disorder.

## CEREBRO-SPINAL FEVER.

While many object to the term cerebro-spinal fever—Stille remarking that it is a name educated physicians ought not to tolerate—it seems less objectionable to the author than other names hitherto proposed. The term cerebro-spinal meningitis does not recognize the infectious character of the malady, and this is an important consideration. The addition of the adjective “epidemic” does not add to correctness, for the disease does not always occur epidemically. I think it better therefore to accept a title which at once recognizes that the disease belongs to the great class of infectious fevers, than to use one indicating simply the inflammatory character of the cerebro-spinal lesion.

**Nomenclature.**—Spotted fever; petechial fever; malignant purpuric fever; and numerous other synonyms.

**Definition.**—Cerebro-spinal fever is a specific infectious fever characterized by an inflammation of the cerebro-spinal envelopes and presenting great variations in its clinical course.

**History.**—This disease, so far as most careful research into the literature of the past can reveal, is apparently of modern development. It is true that an epidemic occurring in Turkey during the sixth century is believed by Richardson to have been cerebro-spinal fever; but by others this is denied, they considering the epidemic in question to have been “the plague.” The first account of the disease was based on an epidemic occurring in Geneva, Switzerland, in 1805. In 1806 it made its first appearance in America, at Medford, Mass. In Europe, it very soon spread to numerous other points, and by 1807 had invaded Bavaria, Holland, Germany and parts of England. In all of these places it appeared about the same time, and apparently without any possibility of having been carried by contagion. A few epidemics occurred in this country up to 1837, when the disease ravaged France, the soldiers in the garrisons being especially affected. In some towns citizens suffered also; in others both the soldiery and the civilians. The French epidemic ceased about 1849. It again invaded America in 1842 and at points far removed from any possibility of importation from Europe. From 1854 to 1860 it invaded Norway and Sweden, and in that time carried off more than 4,000 people. In 1863 it appeared in Philadelphia; and in 1866, in New York City. Since these dates neither city has been entirely free from it. Indeed it might be said that cerebro-spinal fever is now endemic in all our large cities, notably Philadelphia, New York, Chicago, Minneapolis, Detroit, etc. Still, with this widespread prevalence, cases of the disease are comparatively rare excepting during epidemic times.



**Etiology.**—Cerebro-spinal fever may occur either as an epidemic or sporadic disease, especially in the cold months of the year. Cold climates likewise favor its spread; hence it is more commonly met with in the northern portion of the United States and Europe. In tropical countries it is unknown. Moisture also seems to favor its development.

Overcrowding, and all the bad hygienic surroundings that this entails, constitutes a very important predisposing factor. This is shown by the frequency with which troops in barracks, children in crowded schools, and inhabitants of tenement houses, have been attacked. Poor and scanty food, unsanitary surroundings, deficient light, impure air, overexertion, and all depressing mental and physical conditions, favor its development. The relation of bad hygiene to cerebro-spinal fever is well illustrated by the graphic report of Flexner and Barker on the epidemic of 1893 in Lonaconing, Md., and other places in the George's Creek Valley. To use the language of these writers: "The town of Lonaconing is a mining centre in the Allegheny Mountains, and contains some 5,000 inhabitants. The location is peculiar and is more or less of interest from an epidemiological standpoint. The town is situated in a narrow valley and extends well up the sides of the steep hills which enclose it. Through the bottom of the gulch runs a muddy stream known as George's Creek. The mountains rise more or less abruptly from the sides of the creek, and the town may, for our purposes, be divided into two parts: first, that lying in the valley in which are situated the business portion and for the most part the dwellings of the professional and business men of the place; and second, that built on the mountain sides, consisting almost entirely of miners' dwellings. The stream and valley are somewhat tortuous at this spot and give the place the appearance of being surrounded on all sides by mountains.

"One is struck almost at once by the arrangement of the houses on the incline. They are placed in long rows, tier above tier, facing the valley; behind and therefore above the houses are situated the privies, placed flat upon the ground; and not infrequently the cow-stable is located quite close to the dwelling. Before our arrival at Lonaconing there had been a heavy snowfall, but the weather soon afterwards became mild, the snow melted, and exposed to view and made doubly striking by contrast the dreadful conditions which existed. The water streamed down the mountain sides, carrying with it the general refuse from the yards, the material from the cow-stables, and the excreta from the out-houses of the upper tiers, through the yards, past the dwellings situated below, and finally entered the creek, which acts as a huge sewer winding through the centre of the town. On their way these polluted surface washings found no system of drains for their reception, and in places crossed the common roadway in little rivulets, through which the inhabitants of the town had to drive and walk.

"The water supply of Lonaconing is derived partly from surface wells and partly from cisterns, and we actually observed during a thaw the entrance of surface water and the accompanying filth directly into wells which were in daily use for drinking and culinary purposes. Significant too is the fact that in the valley the wells are situated often only a few feet from the creek, and it is certainly not hard to believe that the contaminated water from the creek could penetrate through the soil and become mixed with the water of the wells. The slaughter-houses are in the heart of the town upon the banks of the creek, and the blood and waste material from these find their way into its waters.

"These unsanitary conditions are not confined to Lonaconing alone, but in its course, George's Creek passes through a number of small towns, each of which adds its quota to the general filth, and only a short time ago an investigating party inspecting the river between Westernport and Phoenix Mines found among other things carcasses of horses in the stream or lying upon its banks."

Certainly the above pen-picture shows a frightful condition of sanitation, and it is not surprising that the place should be the location of as severe an epidemic of cerebro-spinal fever as has occurred in recent years. Indeed we wonder that numerous filth diseases were not also epidemic.

While cerebro-spinal fever shows such a decided preference for unsanitary places and for the poorly nourished, it may invade the homes of those living in the midst of the most approved sanitation and eating the best the market affords.

Cerebro-spinal fever is regarded as but feebly, if at all, contagious. Evidence in support of this view is found in the fact that it very rarely happens that more than one case occurs in a household, that physicians and nurses in attendance upon the sick are rarely attacked, and that there is no regular period of incubation after so-called exposure to the contagion. In epidemic times, there is a marked peculiarity in the spread of the disease, a peculiarity that speaks directly against the contagious character of cerebro-spinal fever, *i. e.*, the affection breaks out at numerous foci far away from each other, and without means of intercommunication. In contagious diseases generally, the spread of the epidemic is by peripheral extension, and along the channels of commerce and travel.

Recognizing these facts, we must not close our eyes to certain others which do show that cerebro-spinal fever may sometimes be contagious. Instances in which it has been communicated by fomites, and still others in which the poison has clung to certain dwellings, have been reported, and should teach careful disinfection of the sick-room and its furniture.

Statistics apparently show a predisposition of the male sex to the disease. This is doubtless due to the fact that men are more exposed to the causes which predispose to cerebro-spinal fever, occurring as it does in hospitals and camps. In private practice, however, the cases are about equally divided between the sexes.

Age undoubtedly exerts an important etiological influence. The majority of cases occur in children under five years of age. Attacking infants of less than one year, it is very fatal. No time of life, however, is exempt. Liability to the disease is small after the tenth year, and is almost *nil* after the thirty-fifth.

It is an interesting fact that during the epidemic prevalence of cerebro-spinal fever, meningitis occurs with remarkable frequency as a complication of other diseases.

The occasional tendency of cerebro-spinal fever to attack the lower animals during its epidemic prevalence among the people, is worthy of note. This fact was observed among horses during the Vermont epidemic of 1811, among dogs and pigs, in Ireland, and among horses again in New York, in 1871.

**Pathology and Morbid Anatomy.**—Cerebro-spinal fever must be regarded in the present state of our knowledge as dependent upon a specific microbe. Eberth, Bozzolo, Leyden, Weichselbaum, Fraenkel, Gabbi, Puritz, and more recently Flexner and Barker, have investigated the subject and have discovered in the meningeal exudation a micrococcus, resembling morphologically the diplococcus of pneumonia. Pushkareff, of St. Petersburg, has cultivated this organism, developing a progeny which likewise resembles the pneumococcus. His material, however, was derived from pus found on the meninges of persons who suffered from cerebro-spinal meningitis as a complication of pneumonia. Other bacteriologists investigating this subject have discovered other bacilli than the diplococcus. Thus one New York physician reports his investigations of three cases, the bacteriological results of which were that he discovered different bacilli in each one. His observations, however, must not be regarded as iconoclastic, but rather as part of our accumulating knowledge. Kainen and Adenot discovered in one case a bacillus apparently identical with the bacillus of typhoid fever, and in others numerous streptococci and staphylococci. Then again, Kleppen, in 1892, discovered the micrococcus lanceolatus in the exudate of meningitis arising from traumatism and ear diseases, and occurring in association with other diseases than pneumonia, as inflammation of certain serous cavities. The regularity, then, with which the diplococcus has been found by bacteriologists of acknowledged ability points strongly to it as the essential cause of the disease. The occasional discovery of other micro-organisms shows that these also may be the cause of some cases; and the presence of the lance-shaped coccus in meningitis of traumatic and infective origin leads to a suggestion that this micro-organism does not necessarily produce the special type of disease under consideration.

The manner in which the micro-organism gains entrance to the system is of considerable interest, but has not yet been determined. Its



widespread prevalence in nature is universally acknowledged. It is known that in a large percentage of the healthy it can be found in abundance in the saliva. It has also been observed with considerable interest that cerebro-spinal fever prevails epidemically when certain other diseases, notably pneumonia and parotitis, are of frequent occurrence in the neighborhood. The same bacillus is found in all of these affections. Suggestions to the effect that infection takes place by way of the blood-current, the nasal cavities, or the gastro-intestinal tract, have been offered. The occurrence of cerebro-spinal meningitis, secondary to pneumonia, suggests very strongly the possibility of blood infection in this particular instance. The association of other cases with inflammatory conditions of the nasal cavities and accessory sinuses, has suggested an infection by continuity from these cavities, a suggestion that must be accepted for some cases, but cannot be entertained in others. Flexner and Barker incline to the view that the infection is by way of the gastro-intestinal tract, and observe that in the cases complicated with dysentery, the diplococcus was found in the discharges.

The conclusion regarding the causation of cerebro-spinal fever seems to be that it is a miasmatic disease, the organisms of which retain their virulence for months while still dry, and that they are widely diffused in nature, but become active only when external circumstances and personal predisposition are favorable.

The pathological changes in cerebro-spinal fever vary according to the duration of the case. If the patient succumbs rapidly the cerebro-spinal membranes will be found congested, and nothing more. This condition may be limited or widely diffused. Microscopic examination shows some emigration of leucocytes. Few cases last for many hours, however, without manifesting some degree of exudation, which consists of pus corpuscles, blood cells, fibrin and serum. It takes place into and beneath the pia mater. With protraction of the inflammation the sero-fibrinous matter is exuded in larger amount and contains pus cells in greater or less quantities. In the severe grades of inflammation the surface of the brain may be bathed in pus. The exudation, as is also the hyperæmia, is found greatest in the depression between the convolutions, and notably so in the fissure of Sylvius, about the optic commissure, the pons varolii and the surface of the cerebellum. The arachnoid is often dense and cloudy, especially along the vessels and in the depressions. In prolonged cases, a sero-fibrinous or purulent exudate may be found within its meshes. The cerebral substance itself is usually congested and sometimes inflamed, local or diffused softenings are occasionally observed. The ventricles contain serum in excess, sometimes in sufficient quantity in connection with the effusion on the surface of the brain to produce flattening of the convolutions. Punctate hæmorrhages and cerebral œdema are occasionally noted. Abscess of



the brain has been observed, but it is very rare. In some cases the exudation of serum into the ventricles fails to be absorbed and chronic hydrocephalus follows.

Changes in the blood are present. In the asthenic cases it is found fluid and dark. Occasionally bubbles of gas are found in the heart and arteries. In sthenic cases, or in cases in which pyrexia has not been great, the blood is hyperfibrinous and coagulates readily. White clots are then found in the heart. Ecchymoses are found in the connective tissues of various portions of the body.

The lungs may be found congested or inflamed; bronchitis, oedema pulmonum and atelectasis are other conditions found in the respiratory tract.

The spleen is generally congested and enlarged, the increase in size usually being in direct proportion to the degree of pyrexia. It is, however, never as large as in typhus fever. The kidneys are sometimes found to have taken on the changes characteristic of nephritis.

**Clinical Course.**—The clinical course of cerebro-spinal fever stamps it with the identity of an acute infectious fever. The onset is usually sudden, accompanied by chill, fever, prostration, general pain, and severe headache. Vomiting may occur at this period, or even later, and become obstinate. The intense headache and stiffness of the neck, which rapidly develop, are highly suggestive of the nature of the oncoming illness. The facial expression is altered, the face being usually pale and anxious. The pupils may be dilated, contracted or unequal. In a certain percentage of cases the general pain is intense and greatly aggravated by any motion. Hyperæsthesia with formication may be present, and usually appears first in the lower extremities, but may ultimately involve the entire body. The organs of special sense also share in the general hypersensitiveness, so that the patient complains bitterly of noise, light and jars. The suffering, even from movements incident to nursing, is so great that the patient is in a state of fear of motion. With the lapse of a few days, the stiff neck becomes rigid, or by involvement of lower parts, opisthotonos is developed. Clonic spasms of the muscles of the face and extremities, accompanied or not by violent delirium, are present in well-developed cases. In some cases the *cri hydrocephalique* is present. The temperature is marked by great variability, the early rise being rapid, but subsequent fluctuations, even to below normal, are frequent. Hyperpyrexia may occur just before death. The irregularity stated to occur in the temperature range is manifested also in the pulse and respiration, which present remarkable variations within limited periods of time. The increase in frequency of the pulse-rate is often relatively much greater than the increase in the temperature. The tongue presents the appearances common to the infectious fevers. The bowels are constipated, and the abdomen sunken in a marked degree; this latter

phenomenon being attributed by Traube to contraction of the intestines, the result of irritation of the pneumogastric. The urine is usually scanty; it may even be retained, requiring the use of the catheter, or be discharged with difficulty.

Eruptions of varying character appear with full development of the disease, but none of the varieties are characteristic. In the early stage a scarlatinous redness affecting especially the face and neck may be present, and is followed later by a roseolar eruption upon the limbs and trunk. Exanthems resembling the eruptions of measles, erysipelas, and the vesicular eruptions respectively, have been reported. Petechiæ occur in a minority of cases. Stille met with eruptions in about one-half of his cases, while other observers have failed to observe them in any. Herpes is common, appearing upon the face and neck, and often extending to the trunk. Ziemssen regards them as of trophoneurotic origin.

The great variations displayed by cerebro-spinal fever in its symptomatology have led to numerous classifications into many clinical types, which serve, as a rule, to add confusion to a study of the disease. The simplest is always the best, and I believe that the classification employed by Pepper is the most useful from a practical standpoint. This provides for the ordinary form, which has just been described. Other types are as follows: simple, malignant or foudroyant, abortive and intermittent.

In the *simple* or *mild* type, the symptoms are of the mildest possible kind. They consist, as a rule, of cervico-occipital headache, vertigo, rigidity of the neck, and slight nausea and vomiting. Did they not occur during epidemic periods they would fail of recognition as examples of cerebro-spinal fever. Very often the patient goes about his ordinary duties, not thinking it worth while to consult his physician. This is a great mistake, as these cases may develop without any warning into those of the most severe character.

The *foudroyant* form comes on with the greatest suddenness and malignancy. The patient is almost at once thrown into a state of collapse. The chill is violent; fever is absent; indeed, the temperature may be subnormal. The skin is cold and clammy, respirations are slow and labored, and the urine is scanty and highly albuminous. Spasmodic symptoms are prominent; the rigidity is great, and clonic spasms are general and persistent. The malignancy of the case is seen also in the rash, which is of the purpuric variety, and vesicates or sloughs. Nearly all of these cases tend rapidly to a fatal issue, sometimes within six hours of their inception. In some few instances reaction is established, and then the patient is destined to pass through a long illness characterized by the severity of its symptoms and its dangerous complications, often ending in sequelæ which disable the patient for life.

In the *abortive* form, the symptoms begin as in the ordinary cases,

but soon cease. It is believed that in these the first effects of the disease are expended on the blood, with the appearance of all the early characteristic symptoms. The evidences of the subsequent inflammation of the cerebro-spinal meninges are wanting.

The *intermittent* form is characterized by marked remissions or intermissions of the fever, and all the other symptoms of the disease. The regularity of the intermissions resembles that of pyæmia rather than the typical regularity of malarial intermittents.

**Analysis of the Symptoms.**—The symptoms of cerebro-spinal fever are to be explained partly by the systemic infection, and partly by the cerebro-spinal inflammation. As a rule, prodromata are not marked. Some few cases do not begin suddenly as described in the preceding paragraphs, and in them are observed depression, anorexia and general malaise for two or three days before the onset. The chill often comes on at night while the patient is asleep. In very young children it may be accompanied or replaced by a convulsion. It is apt to be especially severe in cases occurring early during the epidemic period, and is also less apt in such to be attended by prodromic symptoms. Sometimes the chills are repeated at intervals throughout the course of the disease, each chill indicating a fresh extension of the affection.

The degree of fever varies greatly. In the form called *foudroyant*, the rise is rapid, and the temperature may attain a very high point within a day or two. Ordinarily, the temperature rises from 100° F. to 103° F. within the first forty-eight or seventy-two hours. It must be remembered that the height of the fever bears no definite relation to the severity of the case. Thus, fatal cases may present a temperature of at no time greater than 100° F. to 101° F. Increase of fever is not uncommon; repeated observations of increase of temperature after death have been reported. Fluctuations of temperature in this disease are so frequent that it may be said that irregularity is its special feature. Ziemssen and Baumler both state that it is seldom that curves can be found resembling each other. These irregular variations in temperature have been attributed to disturbance of the heat-regulating centre, œdema of the brain, the extension of the cerebro-spinal lesion, and the successive involvement of various organs, *e. g.*, the bronchi, the lungs, the serous membranes, the gastro-intestinal tract, etc. In none of the infectious fevers is the average temperature as low as it is in cerebro-spinal fever, and in none are such remarkable variations manifested. Ziemssen has observed a normal temperature in the presence of severe general and local symptoms, and Hart has observed fluctuations in a single day amounting to six degrees. The maximum temperature is not always found in the evening; on the contrary, it is just as likely to take place at any other period of the twenty-four hours.

The pulse displays as great irregularities as does the temperature.



In the beginning it is often below normal in frequency, herein differing from all the other infectious fevers. Like the temperature it presents remarkable variations, and these may be both as to volume and rhythm, and are observed even in those cases running a favorable course. In others progressing to a fatal issue it frequently becomes weak, thready, intermittent, and even imperceptible.

The pains of cerebro-spinal fever are of the most intense variety. They are most prominent in the head, which may be affected all over, or they may be limited to definite localities. They are invariably present in all cases excepting those rapid ones in which the poison spends its full force on the blood, and which tend rapidly to a fatal issue. The pain is most excruciating in severity, causing the patient to cry out loudly in the midst of delirium or apparent coma. The spine is also affected, the pains here being most severe on either side of the spinous processes, and are greatly aggravated by pressure and movement, and are accompanied by a general hyperæsthesia. This symptom is usually developed first in the lower extremities, extending later to the upper extremities, to the body, and even to the face. The tenderness is frequently sufficient to seriously interfere with proper care and examination of the patient. The special senses are often affected, so that light, sounds and odors become painful.

Tonic spasm of the muscles of the back of the neck is a very common symptom. Ordinarily it may amount only to a simple rigidity. In extreme cases the head is arched backwards, so much so indeed as to interfere seriously with deglutition. It appears usually on about the second day of the fever. When it once appears it persists until the close of the disease. The spinal muscles are sometimes involved in the spasm, and in extreme cases there may be opisthotonos. When the spasm involves the extremities, the usual position is that of flexion of the legs on the thighs, and the drawing up of the thighs on the abdomen. Occasionally trismus is observed. Sometimes the meningeal inflammation is strictly localized, and the spasm is correspondingly limited, arising from the local irritation. In many of such cases a localized paralysis with subsequent atrophy of the affected muscles ensues.

A very interesting symptom has been described by Kernig, of St. Petersburg, who regards it as pathognomonic of meningitis. It consists of a peculiar "flexion-contraction," which cannot be reduced when the patient is in the sitting position. The symptom is also present when the patient lies on his side with his thighs drawn up; but it is absent when lying or standing. Kernig's observation is interesting, but as yet lacks confirmation by others.

Clonic spasms are far less frequently observed than are those of the tonic variety. They may be general or local in distribution, and may consist only of a simple twitching. They may develop in the beginning



or be delayed until late. They may appear but once, or be repeated a number of times over several days, and they may accompany or be succeeded by paralysis, or alternate with tonic spasms.

Paralysis, though of very rare occurrence, is worthy of note as a motor symptom of cerebro-spinal fever. It may be of either the hemiplegic or paraplegic type, or again it is localized to muscles associated functionally or anatomically, or to others supplied by individual cranial nerves. Of the latter we find ptosis and strabismus the most frequent. As a rule, these paralyzes of cerebro-spinal fever are temporary; occasionally they persist through life.

As can be readily surmised from the pathology of the affection, the knee-jerks present no regular condition. Often they are exaggerated, while in other cases they are diminished or abolished. The cutaneous reflexes are unimpaired.

Eye-symptoms are present in great variety. Sometimes they are of mild degree, while at others they are so severe as to amount to veritable complications. The pupils may be either contracted or dilated, oftener the latter; sometimes one pupil is dilated, while the other is contracted. They may also exhibit remarkable and sudden changes in size (hippus). Owing to the pressure of the exudation on the optic tracts, blindness may be an occasional symptom very early in the case. In many instances photophobia is distressing. Strabismus sometimes occurs, and is nearly always of the convergent variety. In the Lonaconing epidemic it is interesting to note that in nearly every case in which strabismus existed, it was divergent. Inflammatory affections of the eye are also observed. Early in the disease conjunctival hyperæmia and inflammation with or without ecchymoses appear. Destructive inflammations of the eye may also set in, and include iritis, suppurative irido-choroiditis, keratitis, etc. Optic neuritis is not uncommon, and may be associated with thrombosis of the retinal veins, as reported by Randolph. In very exceptional cases nystagmus is observed. The frequency of eye-symptoms in cerebro-spinal fever may well be surmised, when it is stated that Randolph found them present in all but one of thirty-five cases examined by him in the Lonaconing epidemic. In the majority of cases, the ocular symptoms disappear, leaving no trace behind them. In many, on the other hand, there results complete destruction of the eyeball, and in those in which optic neuritis is the condition, blindness from secondary atrophy is not uncommon.

The deafness of cerebro-spinal fever may be either late or early. Occurring early, it probably arises from pressure of the exudation on the auditory nerves. Occurring later, it may be due to suppurative inflammation of the labyrinth, or catarrhal inflammation of the middle ear. It is said that when the deafness occurs in both ears simultaneously, it is due to pressure of the exudate on the auditory nerves. Such deafness is

rarely permanent. Permanent deafness is very frequently the result of cerebro-spinal fever, if the statistics of institutions for the deaf are trustworthy. Moos, investigating these cases, found that in more than one-half of them the deafness was associated with disturbances of equilibrium.

Vertigo is an early accompaniment of the headache; it may appear with the prodromic symptoms. It may be either mild or severe, and be present either while walking or reclining. In some cases it is due to disturbances in the labyrinth.

In the majority of cases, the mental condition is one of apathy. In those of more severe type, delirium is present, which, in fatal cases, may go on to coma. The delirium presents all grades and varieties, from slight rambling talk to wild mania. Sometimes it consists of a mere restlessness, and this in mild cases only. Sleeplessness is not uncommon. One form of delirium may change into another. The period at which delirium appears, is usually the second or third day; exceptionally, it may be one of the first symptoms. In fatal cases it may merge into coma.

The gastro-intestinal symptoms are not characteristic of cerebro-spinal fever. Vomiting is often an early symptom, but is cerebral in origin, and does not depend upon gastro-intestinal disturbance. In some cases it is so persistent as to become dangerous by the exhaustion it induces. The tongue is usually moist, and white down the centre and at the tip and edges. It may become dry and cracked. The bowels are usually constipated; diarrhœa is sometimes observed. The abdomen is often as markedly retracted as in cases of tubercular meningitis.

Quite a variety of eruptions may accompany cerebro-spinal fever. Although this feature of the disease is responsible for the name by which it was at one time largely known—spotted fever—it is by no means a constant phenomenon. Many cases run their entire course without the vestige of an eruption. In some, herpes is an early symptom, making its appearance especially about the mouth and lips, and other portions of the face. Petechial and purpuric spots are often present. They may cover the entire skin, or be limited to definite areas, as in one of my cases, to the extensor aspects of the large joints. Other types of eruption observed in cerebro-spinal fever are erythema, ecthyma, urticaria, and pemphigus.

The urine is concentrated as in febrile affections generally. It sometimes contains albumin in small quantities, and then under the microscope, hyaline and granular casts are usually found. An excess of phosphates has been found by Flexner, Barker, and von Grimm.

Many of the cases suffer from joint affections. The knees, elbows, wrists and ankles are especially liable to this complication. They are swollen, the swelling dependent upon both intra-articular and peri-articular effusion. Sometimes they constitute such a prominent feature of

the case as to suggest very strongly the possibility of acute articular rheumatism being the primary disorder with a secondary meningitis.

Splenic enlargement is discoverable by percussion in about one-half of the cases.

**Complications.**—The majority of the complications of cerebro-spinal fever have been referred to in the preceding review of its symptomatology. Pneumonia seems to be its most frequent complication. Very often, meningitis occurs as a secondary condition in pneumonia. In some cases in which the two diseases are present, it may be difficult to decide which is the primary disorder. Pericarditis, endocarditis, and pleurisy, have been observed in some cases.

Permanent changes in the brain structure are sometimes noted. Thus meningeal thickenings may occur and lead to severe headaches which persist for years; in other cases, the same condition may give rise to paralysis, contractures, etc. Sometimes mental impairment ensues, taking the form of poor memory, dementia, or disorders of speech.

**Diagnosis.**—In children cerebro-spinal fever may simulate *pneumonia*, but here physical examination should guard against error. There should be no difficulty in recognizing cases in which the pneumonia complicates cerebro-spinal fever, if the physician is sufficiently alive to his duty to make routine physical explorations of the heart and lungs at every visit.

Osler directs attention to the danger of confounding certain cases of *typhoid fever* with cerebro-spinal fever. The danger lies in not recognizing typhoid cases beginning with delirium, pains in the head, retraction of the neck, high fever, spasm and tremor. He reports such cases which he states would certainly have been regarded as examples of cerebro-spinal fever had not autopsies shown them to be typhoid fever. I have seen cases corroborating this statement.

*Tubercular meningitis* sometimes resembles the epidemic disorder. When doubt exists, the history of the patient should be carefully considered. In tubercular meningitis there is usually a scrofulous diathesis or a history of tubercular inheritance. The development of the disease is generally quite gradual; the pulse is apt to be slow; and the temperature in the early stage, low. Eruptions do not develop, delirium is slight, and muscular rigidities or spasms do not occur until late, and then are not as marked as in cerebro-spinal fever.

*Meningitis* from other causes must sometimes be differentiated from the epidemic disease. Thus in one case seen with Dr. Bartlett, a very important point of diagnosis was called for. The patient had had an obstinate otorrhœa for years, but had persistently neglected it. Cerebral symptoms set in suddenly, but were accompanied by rigidity of the neck and back, and peripheral spinal pains. This led to an opinion that the case was one of cerebro-spinal fever, the diagnosis being confirmed



by a petechial eruption two days later, and the subsequent course of the case.

Severe *variola* may be confounded with cerebro-spinal fever. In smallpox rigidity of the neck-muscles rarely occurs, and if so, only late in the disease. The eruption appears first on the forehead upon the fourth day, and the throat is sore, an important symptom absent in cerebro-spinal fever. The headache in smallpox is frontal, in cerebro-spinal fever it is occipito-cervical.

*Scarlatina* is distinguished especially by the erythematous eruption, the early redness of the fauces, and the strawberry tongue.

Some cases of *pernicious malarial fever* may be confounded with cerebro-spinal fever, when there are coma and rapid development of collapse. The history of the case is here invaluable, as pernicious malarial fever rarely exhibits its malignant character in the first paroxysm. Microscopic examination reveals the presence of the characteristic organisms in the blood.

**Prognosis.**—The mortality varies greatly in different epidemics. According to Hirsch, it will range from 20 to 75 per cent. His remarkable statistics show that in a collection of 15,632 cases the death-rate amounted to 37 per cent. This I am satisfied may be accepted as the average mortality under allopathic treatment. The prominence of severe cerebral symptoms, pupils uninfluenced by light, the youthful character of the patient, a plenteous eruption, a failing circulation, collapsic symptoms, persistent vomiting, embarrassed respiration, or any of the complications detailed, should lead to unfavorable prognosis. Protracted cases present a large mortality, and of the foudroyant cases nearly all die.

Cerebro-spinal fever is remarkable for the prolonged convalescence in many cases.

**Treatment.**—While most authorities, as already stated, do not consider cerebro-spinal fever contagious, others believe it to be so in some degree, and advocate isolation of the patient and the adoption of general methods employed to limit the spread of contagious diseases. As the affection often prevails within restricted areas, it is safer to remove young subjects who are especially susceptible, from the infected district. Crowded and unsanitary tenements should receive proper attention, and the personal hygiene of the inhabitants improved as much as possible. It is better that those in actual attendance upon the sick should employ thorough personal disinfection before associating with outsiders.

Successful treatment must be one capable of arresting cerebro-spinal inflammation, for, as Bristowe remarks, "the mortality of the disease is due less to the direct influences of the specific poison than to the cerebro-spinal inflammation, which is one of the immediate consequences of its operation" (Practice of Medicine, p. 202).

For the frankly inflammatory cases, Hughes thinks *aconite* the most



perfect *simile*. Cases of this character are, however, not common, and aconite is not therefore often used. *Belladonna*, *gelsemium* and *bryonia* have accomplished much more, and must be differentiated by well-known indications. This class of medicines is very quickly outrun by the disease, as it soon manifests cerebral or spinal symptoms, which they are not sufficient to combat. For this stage, of the fully developed cerebro-spinal fever, no remedies, in my experience, are equal in beneficial results to *cuprum aceticum* and *cicuta*. It is proper to say, however, that this experience has been gained almost entirely with sporadic cases, a few of which have been seen in the course of winter months of most years. The former medicine has been administered to most cases, especially if the symptoms were more prominently cerebral. *Cicuta* has been given for the more general symptoms due to spinal involvement. A careful study of the pathogenesis of these medicines will satisfy one of their suitableness for the treatment of this disease. *Cuprum* has been given in most cases as soon as the severe headache has been attended by contracted or unequal pupils, pain, and some rigidity in the back of the neck, etc. If *cuprum* is not efficient in arresting the development of the symptoms growing out of the inflammation of the cerebral meninges, and especially if pains, hyperæsthesia, and spasmodic action of the muscles of the extremities and trunk appear, indicating involvement of the cord, *cicuta* had better be administered if another medicine is not clearly indicated. Experience with this remedy has been quite extensive and satisfactory; some observers being quite enthusiastic in its praise.

In the petechial type, the class of medicines to be recommended is the same as suggested for typhoid fever (see page 76), particularly *arsenic*, *rhus toxicodendron*, *baptisia*, *crotalus*, and *lachesis*.

Dr. W. S. Searle, of Brooklyn, has found *actea racemosa* useful for the pains and spasms which may continue after the acute symptoms have subsided.

*Veratrum viride* has been used in the early stage before exudation has taken place, and apparently with some success.

*Camphor* and *veratrum album* are of value when collapsic symptoms attend the onset of the disease.

When cerebral exudation with coma and an absence of marked spinal symptoms exist, *cuprum*, *apis*, *opium* and *helleborus* are suitable.

*Hyoscyamus*, *arnica*, *physostigma*, *cannabis indica*, *agaricus*, *nux vomica*, *æthusa cynapium*, *zinc* and *cocculus* have also been recommended.

During the progress of this painful and distressing affection, everything possible must be done to alleviate suffering. While I have never administered morphia to one of these cases—a practice altogether too common among our old-school brethren—it may be sometimes necessary. Careful prescribing should not be neglected on this account, however.

The patient is to be carefully fed upon liquid diet from the beginning, and alcoholic stimulants employed if the usual indications for them are present. Rectal feeding may, in extreme circumstances, be necessary. The bladder must be carefully watched, and the catheter used if required. The bowels should be moved by enema or suppository. There are advocates for both hot and cold applications to the spine; the former predominate. Cold should be used with caution. The old school, leech in the early stage, and apply blisters after the acute condition has subsided. Opiates for the control of severe pain, and hot water to the extremities, if cold. Iodide of potash, in quite large doses, is being given much at present, even from an early stage. The continued current through the length of the spine favors absorption of the exudate, and faradism to paretic muscles assists in the preservation of nutrition.

## PNEUMONIC FEVER.

**Nomenclature.**—Pneumonia; croupous pneumonia; fibrinous pneumonia; lobar pneumonia; lung fever, etc.

**Definition.**—An infectious fever associated with the development of a micro-organism, the *diplococcus pneumoniæ*, the specific lesion of which is a peculiar fibrinous or “croupous” inflammation of the lung parenchyma and which is usually confined to one lobe. Secondary infective processes may involve the pleura, the endocardium, the pericardium, and the cerebro-spinal meninges.

**Etiology.**—From time immemorial pneumonia has been considered as the result of the action of cold, hardly a voice being raised in opposition to this view until within recent years. To the influence of those eminent clinicians, Jürgensen in Europe, and Austin Flint in this country, we are especially indebted for the separation of pneumonic fever from the phlegmasiæ, and placing it in its proper position as a member of the acute infectious group; and to Fränkel for the discovery of the organism which is now generally accepted by bacteriologists as its cause. But although acknowledged as infectious in nature, and the specific organism identified by all competent observers, none have yet placed the description of pneumonic fever among those of the infectious group.

**DIPLOCOCCUS PNEUMONIÆ.** As indicated by its name, this organism is found in pairs. It is elliptical in form (often being designated “lance-shaped,”) and is usually encapsuled. If ordinary cover-glass specimens are treated with glacial acetic acid, and then without washing with a drop or two of aniline oil and gentian-violet, the latter being poured off and renewed several times, the diplococcus will be clearly exhibited. In pneumonic fever this parasite is found plentifully in the expectoration, and in nineteen out of twenty cases Monti found it in fluid drawn by means of Koch’s syringe from the hepatized lung. The diplococcus is evidently widely distributed, being found in the nose and accessory cavities, even in the Eustachian tubes. Netter has discovered it in the buccal secretions of about 20 per cent. of healthy persons. The usual point of attack is the lungs, but under certain circumstances it invades other tissues, secondarily developing complicating inflammations especially of the pleura, endocardium, pericardium, and the cerebro-spinal meninges. It is evidently unnecessary for the lungs to be always first attacked, as Fränkel’s germ has been found associated with inflammations of the pleura and meninges independently of primary changes in the lungs. It is probable that the germs leave the body chiefly by the

sputum, retaining their vitality for long periods of time external to the body, and that dessication is not destructive to them. Bozzolo found the pneumococcus in the milk of a woman five days sick of pneumonia, milk pressed from the breast giving rich cultures.

Granting, then, the infectious nature of pneumonia, all other alleged causes must be relegated to the class of *predisposing influences*. General predisposition is a most important factor, although in what it consists we do not know. Of all infectious diseases pneumonic fever recurs with the greatest frequency. It is not uncommon for an individual to have had two, three, or even five or six attacks of pneumonia; and as many as ten or twelve attacks of pneumonia have been reported as occurring in one individual. Statements of this character must be received with some caution, however, as attacks of catarrhal pneumonia, and various inflammatory attacks occurring in the course of phthisis pulmonalis, are often rated as "pneumonia." Undoubtedly the most important predisposing influence is cold. The belief in this agency as an exciting cause of pneumonia, even before the discovery of a specific organism, was rudely shaken by investigations which resulted in demonstrating that pneumonia increased per thousand inhabitants as the equator is approached, the opposite being true of bronchitis, a disease which in its primary form is universally conceded to be readily and most frequently produced by exposure to cold. It is interesting in this connection to note that many recent observers deny that a history of exposure of this character can often be elicited. While we do not acknowledge that cold can produce pneumonic fever, yet it is often a most important factor. The histories of many cases show undoubted exposure to a low temperature prior to the development of the pneumonic fever, but in the light of our bacteriological knowledge we are forced to consider such a factor as merely developing conditions favoring the activity of the specific organism. Some who accept the specific nature of pneumonia, still believe that a few cases of an idiopathic nature occur. Although the disease is more prevalent in hot countries, its prevalence is greater during the cold months of the year, double as many cases occurring in winter as in summer, and more than double as many in spring as in summer. Variability of temperature is an important etiological factor, the rapid alternations in temperature which are characteristic of the spring months largely explaining the greater prevalence of the disease in this season of the year.

Age is a prominent predisposing cause of pneumonic fever, it being very common in young children and in those of advanced years. From puberty to early manhood and womanhood there appears to be less liability; but after the latter period, it is a frequent disease until about the age of forty-five, when another period of comparative immunity is enjoyed. After sixty it is a very prevalent trouble. A very large proportion of all deaths after sixty years are due to pneumonic fever.



Sex exercises very little causative influence, excepting about the middle period of life, when about twice as many men as women fall victims to it. This is probably due to the exposing nature of men's employment favoring the reception of the poison. This point is demonstrated by the fact that women who work at the same exposing occupations as men are afflicted in similar proportions. The disease does not seem to discriminate between the robust and the feeble, and my own impression is that pneumonic fever pursues an especially active course in strong and robust constitutions. The period of recovery from many acute diseases and in those suffering from chronic malaria, measles, erysipelas, smallpox, and from diseased conditions the symptoms of which depend upon the accumulation in the blood of excrementitious matters (Bright's disease, rheumatism, phthisis), all predispose to pneumonic fever. All agents which diminish the vital powers or disturb the nutrition are also predisposing causes. The geographical distribution of the disease is very general, with, however, a preference for certain regions. Altitude has an important causative relationship, the disease being frequent in high altitudes when north and east winds blow. A crowded population is also an important factor of preference.

*Traumatic influences*, such as blows upon the chest, laborious occupation, or strain from heavy lifting, have been frequently followed by the development of pneumonic fever. In these instances the organism has probably been introduced into the lungs prior to the injury (if an acute one), or the specific agent being at hand (in the secretions of the nose or mouth, and possibly in the bronchial mucus), attacks the lung structure after its nutrition, and probably its circulation, have been disturbed by the traumatism.

The present state of opinion regarding the etiology of pneumonic fever may be briefly summarized as follows: (1) pneumonic fever is a specific infectious fever and due therefore to the influence of a micro-organism; (2) the organism found associated with the great majority of cases is the *diplococcus pneumoniae*. (Weichselbaum considers the distinction of lobar and lobular pneumonia as anatomical only, not etiological.) The relation of other micro-organisms to the disease is not determined, although many hold the opinion that pneumonic fever is not invariably the result of the action of a single organism. (3) The entrance and activity of the micro-organism is probably dependent upon a previous lesion, particularly to the respiratory mucous membrane, such a lesion being in the nature of a catarrh, the inhalation of irritating gases or substances, and in association with these injurious influences, disturbed nutrition, resulting from unsanitary and unhygienic surroundings exercise an important predisposing influence.

**Pathology and Morbid Anatomy.**—The specific lesion of pneumonic fever is located in the lung. The anatomical characters of this

lesion are well-defined and present marked uniformity. The changes consist essentially in morbid processes eventuating in the consolidation of the vesicular structure of the affected organ. In the study of this lesion we are able to distinguish three stages, which have been clearly described by authors, but which have been variously characterized. We will adopt the following as the simplest: (1) *The stage of congestion*; (2) *the stage of consolidation*; (3) *the stage of resolution or diffuse suppuration*.

During the *first stage*, the portion of lung involved is more or less intensely congested, the microscope showing the capillaries of the alveolar walls to be dilated and tortuous, and minute points of hæmorrhage are found especially in the interlobular and subpleural connective tissues. In this stage air still enters the lung. With the progress of the congestive stage the lung becomes heavier, and passes from a reddish to a reddish-brown or purplish color; it pits and crepitates on pressure, but a fragment of it thrown into water will still float. From the cut surface of the organ blood-stained frothy serum exudes, especially if the tissue be slightly compressed. Microscopic examination of this fluid will exhibit swollen granular epithelial cells, which have exfoliated from the walls of the alveoli, and blood corpuscles, both red and white.

In the *second stage*, the stage of consolidation, the air vesicles become fully expanded and occluded by a fibrinous exudate, giving the affected tissue a liver-like character, a condition which has given this stage one of its names—that of red hepatization. The use of this term is not strictly correct, for while the lung has much the consistence of liver, it is frequently paler than normal, owing to an anæmic condition resulting from the pressure of the exudate upon the capillaries of the alveolar walls. If we now compress a small portion it fails to crepitate, is much softened, easily torn, and if thrown into water, sinks. Examination of the cut surface reveals a well-marked granular appearance, the granulations being the little masses of coagulated exudate which are contained in the alveoli and infundibuli, and which become prominent after section of the lung on account of retraction of the tissue after cutting. Scrapings from the cut surface examined by the microscope reveal numerous moulds of the air spaces. The reddish color of the lung which is present in the majority of cases during this stage, is due to the distended bloodvessels of the part, but also partially to the number of red corpuscles existing in the exudate. Microscopic examination of the exudate shows it to consist of the following elements, which vary considerably in their relative proportions in different cases: coagulable fibrin, which contains within its meshes red blood corpuscles, leucocytes and alveolar epithelium. When the fibrin exists in proportionately large quantity it presents tree-like ramifications, giving a characteristic appearance to the microscopic field. At times the red blood cells are found almost to the exclusion of

the other elements, and again the epithelial cells may predominate. The reasons for the special preponderance of one or the other of these elements has not been clearly determined.

The *third stage* is that of resolution or diffuse suppuration or gray hepatization. In this condition the lung preserves many of the characteristics of the second stage, especially those of increased weight, crepitation on pressure, and liver-like consistence; but as the term gray hepatization indicates, there is a change in color from a reddish to a grayish or yellow color. This is largely due to the pressure of the exudate, the destruction of red blood corpuscles within the alveoli, and the presence of large numbers of leucocytes. The lung grows softer with the progress of this stage, the reasons for this being patent upon examination of the tissue. In the first place there is a liquefaction of the fibrin, and therefore a disappearance of the firm fibrinous coagula. The red blood corpuscles, no matter how large their numbers may have been, also disappear before the invasion of the destructive leucocytes. The alveolar walls are infiltrated with leucocytes.

The duration of the several stages varies in individual cases. Only an approximate statement concerning their length can be made. The first stage is seldom protracted beyond the second day of the disease, the second stage lasts from four to eight days or more, and the third stage from two days to two weeks. The transition from the one stage to the other is often rapid, especially in the young; but in the aged it may be still more so, for signs of consolidation are sometimes detected at the close of the first day, and the third stage with many of its unfavorable sequences may develop within the succeeding two days. A gradual extension, by continuity, may be sometimes observed, and such cases have been designated "wandering pneumonia." In this form the various areas of consolidation exhibit, on post-mortem examination, a variety of conditions varying from the hyperæmia and beginning exudation of the first stage, to gray hepatization, or destructive changes; extension of the lesion is not always by continuity, but may be "by leaps."

In cases that do not pass on to gray hepatization, resolution occurs. Whether or not resolution is possible after a well-established third stage has developed, is problematical. It may occur in all probability even during the stage of congestion. This process is pathologically much like resolution occurring in any inflamed part. Much discussion over the disposal of the alveolar contents has taken place. Some hold that they are expectorated after having undergone certain changes, and others that the exudation is removed by the absorbents. It is probable that it is removed in both ways. We know from careful observation that expectoration of the alveolar contents does take place; and we are forced to believe that they are absorbed partially or entirely in certain instances, for the reason that cases of pneumonic fever with extensive consolidation



of lung tissue, pass to a favorable termination with little or no expectoration.

The occurrence of circumscribed suppuration or abscess of the lung in pneumonic fever must be very unusual, for many question whether it ever occurs, many reported cases being obscure. Like abscess, gangrene of the lung is rare. The association of pleurisy with pneumonic fever is so constant as to lead many to designate the disease a pleuropneumonia. The existence or non-existence of pleurisy with pneumonia depends mainly upon the location of the lung lesion. If the latter is centrally located, pleurisy is infrequent. If it extends to the pulmonary periphery, the enveloping serous membrane becomes inflamed, the pleurisy thus seeming to be due to simple extension of inflammation.

The bronchial glands present inflammatory changes which may proceed to suppuration.

The pulmonary vessels supplying the affected area of lung tissue are occasionally thrombotic. The lymphatics of the lung are filled with exudate similar to that contained within the alveoli.

The blood shows slight changes other than an increase of fibrin elements, which increases with the progress of the disease. On account of the short duration of the disease anæmia is never a marked feature. The red corpuscles are diminished, but present little morphological change. The white corpuscles may be doubled in number with return to normal after the subsidence of the fever. The absence of leucocytosis during the height of the disease is undoubtedly an unfavorable condition. Diplococci are seldom demonstrable in the blood.

The most common cardiac change discovered is endocarditis, pericarditis being rather less common. Recent observations show both endo- and pericarditis to be very much more frequent complications than has been supposed. The majority of cases are undoubtedly overlooked, even in the post-mortem room, for until recently it has not been thought necessary to examine the heart in all cases. The right heart often contains coagula, which are soft and pale and which may extend for considerable distances into the bloodvessels. The myocardium is frequently discovered in a state of fatty degeneration. The parenchymatous change is a quite constant feature. The heart cavities may contain thrombi, but the formation of cardiac thrombi before death appears to be rare. Obstruction of vessels at a distance from the heart may result from the same cause, the writer having observed resulting gangrene of both lower extremities. It is needless to say the case terminated fatally.

A slight degree of catarrhal inflammation of the gastro-intestinal tract is not uncommon. Instances have been reported in which there was extended ulceration of the gut with free hæmorrhages. It is questionable if these symptoms are not due to complicating affections, for most such cases have been observed during the prevalence of other diseases.



such as cholera and typhoid fever. Croupous (or diphtheritic) inflammation occurs in the stomach and intestines in a small percentage of cases.

Besides the less common features of the disease there may be changes in the liver, spleen, brain and other organs, similar in character to those attending the acute infectious diseases, *i. e.*, congestion, softening, parenchymatous degeneration, etc.

Meningitis is an occasional complication and is usually confined to

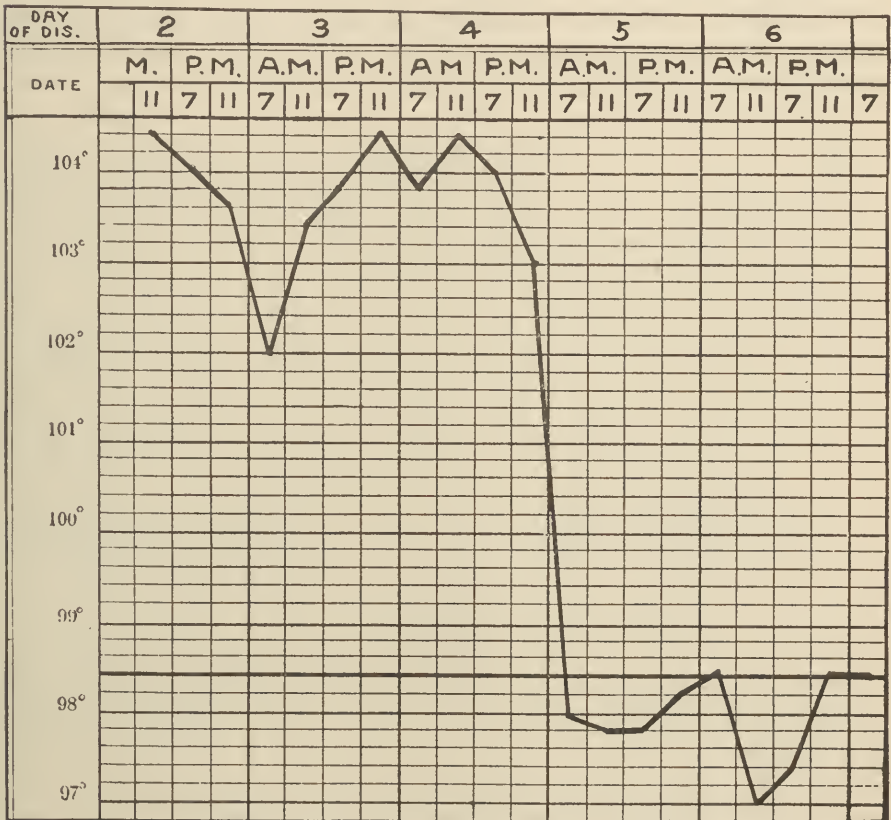


FIG. 7.—TEMPERATURE CHART OF CASE OF ACUTE LOBAR PNEUMONIA.

the convexity. It has been frequently met in association with destructive endocarditis.

The pathological changes are often very limited when grave constitutional conditions are present; this want of parallelism between the extent of the lesion and the general symptoms constituting one of the strongest evidences of the infectious nature of the disease.

**Symptomatology.**—**PRODROMIC PERIOD.** A small percentage of cases of pneumonic fever presents prodromic symptoms, consisting of a feeling of malaise, some oppression of the chest, slight elevation of tem-

perature, a little chilliness, and sometimes slight jaundice. Such a group of symptoms is probably attendant upon a protracted first stage, of mild intensity. Prodromic symptoms of a more general character I have witnessed existing for a week or more prior to the onset.

INVASION PERIOD. As observed in the great majority of instances, the onset of pneumonic fever is sudden, *i. e.*, without warning. Nine-tenths of all such cases begin with a chill, often severe, which may be repeated in the course of the first day. In young subjects, it is not rare for convulsions or gastro-intestinal symptoms to replace the chill. The subsequent rise of temperature is rapid. While a high point is usually reached during the first twenty-four hours, the acme may not be

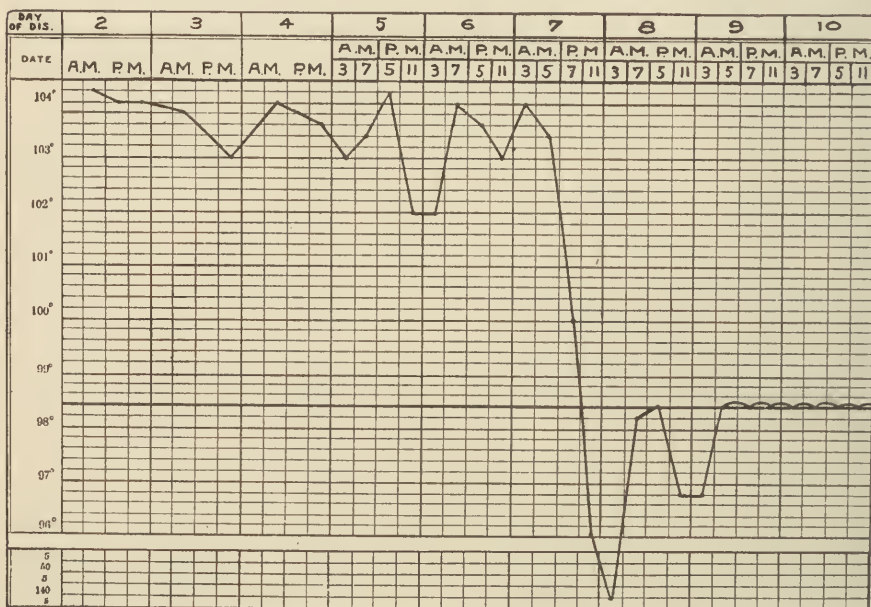


FIG. 8.—TEMPERATURE CHART OF ACUTE LOBAR PNEUMONIA IN AN ALCOHOL HABITUÉ.

THE LOWEST TEMPERATURE REACHED WAS 95° LOWER THAN THE  
HAHNEMANN HOSPITAL CHARTS ARE REGISTERED.

attained before the second or third day, or, in some instances, towards the close of the attack. Sudden elevations of temperature, after an attack of pneumonia appears to be fully developed, suggest the involvement of a new focus, or rapid increase of the primary one. Complications also may lead to such a rise. While in general a close relationship exists between the height of the temperature and the seriousness of the attack, this is not always the case, pneumonias which have terminated fatally having been attended by a low temperature range. A few cases have been reported as running their course without any rise in temperature. Apical pneumonias are usually attended by a higher temperature

range than are others. There are daily remissions varying from one to three or more degrees. The maximum point is generally attained late in the day, remission taking place in the morning. The duration of the fever is very variable, defervescence occurring as early as the second day or as late as the third week. It is most frequent about the seventh or eighth day. In quite a percentage the fall begins upon the fifth, and in a rather less number upon the ninth day. The rapidity of the temperature fall is a pronounced feature of pneumonia. In the majority of cases the temperature falls rapidly to, or below, the normal point. The time occupied varies from four to six or twelve hours, or this may be extended to twenty-four or forty-eight hours. Occasionally a very rapid fall is attended by symptoms of collapse, which may be alarming in degree. Under these circumstances the temperature may fall to  $96^{\circ}$  or  $95^{\circ}$  F., or even much lower. Such a rapid descent in temperature may be attended by several critical phenomena, such as profuse sweating or urination, vomiting, diarrhœa, or hæmorrhage from various orifices of the body. In a few cases defervescence is gradual, extending over a period of several days. This has been especially true of many of the pneumonias we have met within recent years, *i. e.*, since the prevalence of influenza. A rapid fall of temperature followed by an elevation (pseudocrisis) may occur as early as the third to the fifth day. According to Parkes a rise to  $101^{\circ}$  or  $102^{\circ}$  F., subsequently to crisis, with chilliness (a hectic type), may be due to contamination of the blood by the exudate which is undergoing rapid absorption. A marked elevation of the temperature several days after complete defervescence, or the return of the temperature to near the normal point, suggests involvement of another lobe, practically a fresh pneumonia; or the occurrence of pleurisy, empyema, abscess or gangrene of the lung. The chill of pneumonic fever is characterized by its severity, old or feeble persons being often seriously prostrated. So great is the shock to some that collapsic symptoms with unconsciousness may result. With the onset of the fever symptoms there are severe headache, pain in the back and extremities, a short dry cough, rapid respirations, and a stitch in the side. The eyes are bright, and cheeks flushed, the nares active, the expression anxious. Vomiting is quite common in this early stage, and the skin may be slightly icteric. With the progress of the disease the cough is attended by expectoration which is exceedingly tenacious in character, and in most cases contains blood. The intimate admixture of the blood with the sputum leads to the well-known "rusty" color. Rarely, blood may be freely expectorated, and in a much larger proportion of cases it may be nearly or quite absent; the latter is especially true of the pneumonias occurring in the extremes of life. The expectoration which during the greater portion of a pneumonic attack is so tenacious in character that the sputum cup may be inverted without spilling it, towards the



close of the disease is more liquid in character, and consequently more easily expectorated. Expectorations may be entirely absent in children, also in the old or debilitated. The same may be said of cough and pain. The so-called "prune-juice" sputum, which is a dark brownish fluid expectoration, is indicative of a low type of the disease. With the occurrence of defervescence the cough and sputum may quickly disappear, or a muco-purulent material may be expectorated for some time. Examination of the sputum with the microscope reveals a fibrinous mass containing blood corpuscles variously degenerated, epithelium from the bronchi and pulmonary alveoli, fibrinous moulds of the bronchioles, and the characteristic diplococci.

THE RESPIRATIONS in pneumonia are relatively very rapid, *i. e.*, in relation to the pulse-rate, a phenomenon of value in diagnosis. In active cases they are found from 38 to 40 per minute with a pulse-rate of 100 to 115 or 120; rates of 60 to 80 are not rare. The character of the respiration is quite typical, inspiratory efforts being short, and expiration often completed with a little grunting effort. If the chest is inspected the respiratory movements are observed to be limited upon the affected side. This limitation of movement is largely voluntary, and for the purpose of preventing pain; it is therefore a more prominent feature in cases attended by pleurisy.

PAIN is often a most distressing symptom, and is due to the attending pleurisy. There are several foci of pain, one or more of which may be present. The most frequent one is located below the nipple of the affected side, and less frequently, the axillary region or the scapular behind. Occasionally the pain is referred to the abdomen or flank. It is aggravated by all efforts which involve the chest.

THE PULSE, in ordinary favorable cases, is from 100 to 110 per minute. Rates of 120 to 130 are a sufficient cause for apprehension. In serious cases the pulse may be very rapid, feeble, and irregular. The tendency to failure of the circulation in pneumonia is strong, indeed this is a common cause of death. The nature of the failure has not been clearly demonstrated, but it appears highly probable that it is the result of the influence of the poison elaborated by the specific bacteria. Evidences of failure may appear as early as the third day, or as late as the period of defervescence. I observed a fatal case upon the third day, apparently from failure of the systemic circulation. The signs of heart-failure are developed rapidly or slowly. The state of the pulmonary circulation may be determined by the character of the pulmonary second sound, which grows feebler as the right heart becomes less able to empty itself. In connection with this sound it must be remembered that the obstruction existing in the pulmonary circuit in pneumonia produces abnormal accentuation. Occasionally a well-marked acute dilatation of the right heart may be shown to exist. Gradual elimination of the mus-



cular element of the first sound and a shortening of the long pause, are, in connection with the enfeeblement of the pulmonic sound, the most important signs of heart-failure in pneumonia.

**GASTRO-INTESTINAL SYMPTOMS.** Vomiting is quite common in the early stage, especially in children. The tongue is dry and coated, and if a typhoid state develops, may present the characteristic features common to that condition. Constipation is the rule, but epidemics have been observed in which diarrhœa was frequently met. The spleen and less frequently the liver may be sufficiently enlarged to be detected by physical signs. Swelling of the latter organ is largely dependent upon over-distension of the right heart.

**THE URINE** presents one characteristic feature, *i. e.*, great diminution, or disappearance of the chlorides. This is readily determined by adding to a specimen of the urine a few drops of nitric acid for the purpose of holding the phosphates in solution, and secondly, a like quantity of a solution of nitrate of silver, when if chlorides are present they will appear as a white precipitate of chloride of silver. The reappearance of the chlorides in the urine is always a favorable indication. This test should be applied daily. The amount of urine is reduced, is reddish in color, markedly acid, and of high specific gravity. The urea and uric acid are increased. A small quantity of albumin and occasionally casts and other evidences of a nephritic process are often detectable. If icterus is present, bile is found in the urine.

**NERVOUS SYMPTOMS.** Reference has been made to the general pains common to the early stage, these, unless it be the headache, usually quickly disappear. Convulsions are common in children, and less frequently a comatose state. I have witnessed unconsciousness follow the chill and continue for two days, the subsequent course of the case being favorable; also a comatose state developing later in the disease continuing several days and followed by recovery. In adults there is ordinarily an absence of delirium, the mind remaining clear throughout the disease; but in typhoid cases, in persons possessing unsound kidneys, or in drunkards, delirium may be an important feature. The delirium is occasionally maniacal, with attempts at escape or injury and occasionally may be followed by coma. This group of symptoms is always suggestive of a complicating meningitis, but as under these circumstances meningitis rarely attacks the base, exerting pressure upon nerve trunks, a diagnosis is usually impossible. In those who abuse alcoholics the symptoms of delirium tremens may be promptly developed, so early indeed as to obscure the nature of the disease. Mental aberration may continue through the period of convalescence and become a permanent condition.

**SURFACE OF THE BODY.** A herpetic eruption is very common in pneumonia; in some epidemics it has been observed in one-half of the cases. Its most frequent site is the lips, less frequently upon other por-

tions of the face or upon the genitals. Many novel theories as to the relationship of herpes to pneumonia have been advanced. Profuse sweating is an occasional accompaniment of the crisis and is quite often a symptom of great importance during the course of serious cases. It is not uncommon for the patient to have "gushes" of sweat, *i.e.*, attacks of sudden outpouring of sweat followed within a short period by dry hot skin, such sweating attacks occurring repeatedly during the twenty-four hours and adding greatly to the prostration of the patient. The cheeks are usually flushed, and it is not rare to find a bright redness of the cheek corresponding to the affected lung.

**Physical Signs.**—A careful consideration of the pathological conditions peculiar to pneumonic fever, and their progressive development, is essential to a comprehension of the physical signs.

**FIRST STAGE.** During the first stage the respiratory movements upon the affected side are limited largely in proportion to the degree of pain present. There is slight percussion dulness, which gradually increases with the progress of the disease. Upon auscultation the respiratory sound is observed to be weakened over the affected area, or it is harsh, this broncho-vesicular character being usually more pronounced if the breathing is forced. There may be some subcrepitant râles due to inflammatory matter or serum in the finer tubes. The most characteristic auscultatory sign is the *crepitant râle*. It has long been considered the most important physical sign of pneumonia, and especially important on account of its early appearance. Its production has been generally attributed to the separation of the sticky walls of the pulmonary alveoli during inspiration, and to this explanation I must still give my adherence, rather than to the theory of its origin in friction of the roughened pleural surfaces. This sign is evolved towards the close of each inspiratory effort is constant, and is more pronounced after coughing or during deep inspirations. As the pulmonary vesicles fill with the exudate the crepitant râle is eliminated, but reappears with the entrance of air again into these cells. In a small percentage of cases, comprising mostly young and old subjects, this râle may be absent. Occasionally, particularly in cases of pneumonia associated with influenza, and in elderly patients, generally distributed coarse râles may be present. The vocal signs of consolidation gradually develop during the first stage.

**SECOND STAGE.** The limitation of movement upon the affected side present in the first stage and mainly the result of pain, will be found to be more marked in the second stage, its increasing prominence being the result of the development of a solidified, incompressible area. The movements of the normal side are exaggerated. Percussion yields a dull note, which with complete consolidation becomes flat. There is a sense of resistance attending the stroke. At times there is a tympanitic quality, especially anteriorly, or this quality of sound may be simply

developed around the inflamed area. With complete consolidation of the lung, and obstructed bronchi, well-marked bronchial breathing and broncophony will be present. In no disease is bronchial respiration so perfectly developed. It may accompany one or both of the respiratory acts, usually inspiration first and later expiration. The hand applied to the chest will perceive increased vocal fremitus. If the pleural sac contains fluid broncophony is lessened or absent below its upper level, and at this point egophony may be developed. The heart sounds may often be heard in greater intensity over the consolidated lobe. Pleural effusion may sometimes obscure the physical signs. In pneumonia of a lower lobe it is not uncommon to detect amphoric respiration in the upper portion of the lung, especially just below the clavicle. A cracked-pot sound may sometimes be elicited in the neighborhood of a consolidated area, but more frequently over the pneumonic centre, and is then due to the sudden expulsion of air from a large bronchus. The production of this sign is favored by thin elastic parietes.

**THIRD STAGE.** If the third stage takes the form of resolution there is a disappearance of the physical signs in a retrogressive order. The bronchial breathing gives way to a broncho-vesicular quality and broncophony to simple increased vocal resonance; the crepitant râle may again be heard, but is obscured by coarser moist sounds—the “râle redux” of the French. Occasionally the bronchial râles of the resolution period are, as described by Skoda, “consonant.” The physical signs of consolidation may in some degree persist for weeks. If the progress of a pneumonia is unfavorable the third stage becomes a stage of purulent infiltration rather than one of resolution.

**Pneumonia in Children.**—The pneumonia of children presents certain peculiarities which demand consideration. The prominence of nervous symptoms is the most important characteristic, occurring in about one-quarter of the cases. Headache is quite constant, at least in those old enough to complain of it. Delirium is common, being most pronounced at the period of full development, and may be of a wild character. Convulsions occur about once in twelve or fifteen cases, are general in character, and may be repeated several times. They may appear in connection with the initial group of symptoms, or towards the close of the disease, and are frequently followed by a drowsy condition or stupor, less frequently by delirium. They seldom occur after the second year. In connection with these symptoms the pupils may be contracted or dilated, the head may be tossed about, the urine and feces passed involuntarily, the abdomen retracted, and the pulse frequent and often irregular; vomiting is common to all cases. Cough is usually prominent, but may be practically absent during the early days of the attack. The respiration is very rapid, a rate of 50 to 60 or 70 respirations per minute being not at all uncommon. The disturbed



pulse-respiration ratio stated to exist in adults is even more marked in children. The pulse is rapid, may be intermitting, and the Cheyne-Stokes variety sometimes present, but is not at all common. Quite extensive statistics fail to support the statements of the books that apical pneumonia is oftener attended by cerebral symptoms.

The prominence of nervous symptoms in the pneumonia of children, often before the development of cough, and of other symptoms directing attention to the chest, results in a good deal of diagnostic confusion, which is to be avoided chiefly by means of daily careful physical examinations of the chest; but in cases of slight scattered or central consolidations, even physical exploration gives little assistance until the disease is well developed. The affections for which pneumonia in children may be mistaken are especially simple meningitis, cerebro-spinal fever, and tubercular meningitis.

The prominence of nervous symptoms is undoubtedly due to the peculiar susceptibility of the nervous system of the child. While the presence and persistence of aggravated nervous symptoms is unfavorable, the occurrence of a convulsion or mild degree of delirium does not necessitate a less favorable prognosis. Those occurring towards the close of the disease, however, usually precede a fatal termination. In this form the urine should always be examined.

From meningitis pneumonia is separated by its more continued high temperature, the milder character of the cerebral symptoms, the absence of localized paralysis, the presence of the patellar reflex, and the evidences of pulmonary consolidation.

**Clinical Groups.**—The regular form is represented by the ordinary typical pneumonic fever without predominance of any special feature.

**THE EPIDEMIC FORM.** The occurrence of pneumonic fever as an epidemic has long been recognized, but most positive evidence of its not infrequent prevalence in this form has been rapidly accumulating during recent years. A peculiarity of this form is the frequency with which it has been noted as a house epidemic, *i. e.*, attacking a number of persons in a single habitation. A large number of instances could be cited from the periodical medical literature of the past ten years in which three, four or five persons have been attacked in a single family, and upon a more extensive scale in asylums, hospitals, etc. As many as one in seven in prison population of many hundreds has been known to be attacked. The epidemic form varies much as to the prominence of various groups of symptoms. In some epidemics typhoid symptoms are pre-eminent, in others heart complications; jaundice in others, and again, gastro-intestinal conditions may attract most attention. The mortality also varies much.

The group dependent upon *influenza*, as studied during recent years, is often insidious in onset; it is not quite typical in certain features of



its morbid change, and presents a rather high mortality, especially in those past middle life.

The forms developing secondarily to cardiac affections, Bright's disease, diabetes, etc., are usually severe and rapid in their course, and are liable to be accompanied by pulmonary œdema, and terminate in suppuration or gangrene.

When occurring in drunkards obscurity marks the early stage of pneumonia, and often the entire course, the most prominent symptoms being those of delirium tremens. There is a strong tendency in pneumonia of this character to a low range of temperature and a latency of many of the symptoms. A history of chronic alcoholism warrants an unfavorable prognosis.

In the aged there is also latency of symptoms, the true gravity of cases being often overlooked for this reason. Pain, cough and expectoration are moderate and sometimes practically absent; the temperature ranges low, and the nervous and general symptoms are apparently disproportionate to the lesion. When the patient is at rest the respiration may not be accelerated and the sufferer makes no complaints.

The terms typhoid, bilious, adynamic, rheumatic, malarial, etc., as applied to pneumonia, explain themselves. The most important member of this group is the *typhoid pneumonia*, which has been sufficiently commented upon in the foregoing pages.

The *rheumatic form* is characterized by changes in the joints resembling those of rheumatic fever. This feature may appear at any time after full development of the pneumonia.

"*Central pneumonia*" indicates a lesion beginning in the centre of a lobe, and which may extend so slowly that the physical signs of consolidation are not present until the attack is far spent, or before the crisis has occurred. The general symptoms may be typical, even severe; the rusty sputum, disturbed pulse-respiration rate and others of the most important symptoms may be present, yet physical signs of consolidation be absent. By the fifth or sixth day in such a case the crepitant râle may appear, a slight friction sound, some dulness upon percussion, and from this time on the signs of consolidation may rapidly develop. Microscopic examination of the sputum for the diplococcus should be made in doubtful cases.

*Double pneumonia* is rare, and differs from unilateral pneumonia in seriousness only. Many cases reported as double pneumonia, upon clinical evidence only, are undoubtedly unilateral with bronchitis, œdema, etc., upon the opposite side. *Relapse* must be exceedingly rare, many authors stating they have never met an example.

**Complications and Sequelæ.**—PLEURISY. Many, but not all of the complications of pneumonic fever, are due to the invasion of the organism by the parasite; one of the most prominent of these is pleurisy.

The advent of the pleuritic inflammation varies in different cases, usually appearing with the extension of the inflammation to the surface of the lung, or it may constitute the most prominent early feature of the case. When pleurisy appears thus early and constitutes the salient clinical feature, it is designated pleuro-pneumonia. The exudate under these circumstances is often copious and seems particularly rich in fibrin. In the milder pleurisies occurring by direct extension from the inflamed lobe, the exudation is often fibrinous only. A remarkable feature sometimes met, is the development of pleurisy upon the side opposite to the pneumonic lung, but how infection occurs in these cases has not been demonstrated. A purulent accumulation is not unusual, indeed recent investigations have demonstrated a frequency which was unsuspected. Many cases of pleural effusion remain undetected until the period of defervescence, the incompleteness of which event may call for more careful physical investigation of the chest, and the discovery of the accumulated fluid. This condition is marked by continued weakness, usually by some degree of dyspnœa, and if the fluid is purulent, by continued irregular elevation of temperature, although with simple effusion a considerable rise may persist. The pneumonic origin of pleurisies, apparently of the acute general variety, I have been able to demonstrate by means of the microscope, the pneumococcus being present in large numbers. The physical signs are those of pleurisy with effusion, but if the lung is still consolidated there may be heard below the upper level of the effusion broncophony and bronchial breathing. If upon the left side, the heart will be displaced if the collection is a large one.

A GENERAL BRONCHITIS of sufficient severity to prove a serious complication may coexist with pneumonic fever. Often, as in influenza, it may precede the pneumonic development. In a few cases the finer tubes may become involved, this latter feature often being quite general; the physical signs of course are bi-lateral. In general, patients with this complication have a higher range of temperature, their breathing is more difficult, and the right heart more oppressed with consequent higher degree of venous stasis.

ŒDEMA OF THE LUNGS in its general severe type is fortunately one of the rarer complications; while it may occur in the robust it is more often met in feeble individuals who have been undermined by some chronic affection such as diabetes or Bright's disease. It is indicated by the development of small-sized bubbling râles, a copious, frothy, watery expectoration, and the rapid supervention of cyanosis, oppression of breathing, anxiety, and finally coma.

ENDOCARDITIS is the most frequent of the cardiac complications in pneumonia, as shown by the statistics of Osler, contained in his Guls-tonian lectures for 1885. He there states that in 209 cases of malignant endocarditis collected from various sources, 54 cases occurred in

association with pneumonic fever. Not only is endocarditis the most frequent cardiac complication of pneumonia, but it occurs more frequently with this than with any other affection. It is very apt to be developed upon a pre-existing valvular disease. The occurrence of endocarditis may be suspected if septic symptoms appear, or embolism occurs. It also extends the period of fever and disturbs its type. It is, however, true that many of these cases are not manifested by symptoms of a characteristic nature. As in primary endocarditis, the lesion develops far more frequently in the left heart. Meningitis has been frequently observed in association with the endocardial complication. In the majority of cases physical diagnosis yields unsatisfactory results, murmurs being absent.

PERICARDITIS. Acute pericarditis constitutes a rather frequent and serious complication of pneumonia. It seems to be rather more frequent in the extensive pneumonias of the young. Considerable serous effusion may take place, but in the majority the exudation is essentially fibrinous. Symptoms referable to the cardiac region may be absent, or pain, rapid respiration, and a feeble and accelerated pulse may be present; after the heart is considerably oppressed by the exudation cyanosis appears. Occasionally pericarditis may set in early and assume such a degree of prominence as to obscure the co-existing pneumonia. The diagnosis of this condition is ordinarily easy if the præcordial region is carefully explored.

MENINGITIS, as in the case of cardiac inflammations, is a more frequent feature of pneumonia than has been taught, as many as 5 to 8 per cent. of fatal cases having been reported as manifesting this complication. The much greater frequency of the involvement of the convexity accounts for the frequency with which it is overlooked in life. Involvement of, or extension to the base, gives rise to the characteristic features of meningitis and cannot escape detection. The time of development varies—its usual appearance is with the height of the pneumonia—but, exceptionally, it may constitute a feature of the early days or appear late. The earlier the meningitis appears the more likely is it to be overlooked. Embolism has been repeatedly noticed in connection with the meningitis, and both with destructive endocarditis. Cases of pneumonia associated with meningitis are exceedingly fatal. Meningitis complicating pneumonia has been more often observed when cerebro-spinal fever was prevalent, *e. g.*, Immerman and Heller observed nine cases of meningitis complicating thirty cases of pneumonia. An epidemic of cerebro-spinal fever was prevailing at the time.

PARALYSIS as a sequel of pneumonia has been carefully studied by Boullocke who divides it into two groups: one developing during the early course of pneumonia, the second in the late stages. The type may be hemiplegic, or attacking a single extremity or region of the body. It occurs in the young as well as the old, and may mask the lung affec-



tion sufficiently for the latter to be discovered only upon post-mortem examination. In seven out of seventeen cases of hemiplegia collected by Boullocke, no pathological condition could be detected after death to account for the paralysis. Stephen has called especial attention to the high mortality occurring in these cases, nine out of twenty-five patients dying; also to the adynamic character of the pneumonia, in which the brain symptoms had predominated.

JAUNDICE may develop in the course of pneumonia, and apparently bears no relation to the severity of the case. It is more prevalent in some epidemics, and also in certain regions of country. It may depend upon obstructive catarrh, but most cases cannot be accounted for in this manner. Albumin and casts are commonly present in the urine of serious cases, but a well-defined nephritis is rare. It is very unusual for nephritic changes to persist after convalescence. If the kidneys are impaired by chronic disease the outlook is a gloomy one. Many cases of chronic interstitial nephritis are first detected during the course of, or subsequent to, pneumonia. The parotid gland may inflame and suppurate. This complication has been observed in association with endocardial inflammation and is probably infective in character.

**Diagnosis.**—A diagnosis of pneumonic fever can be made only upon the combined symptoms and physical signs of this disease. While the symptoms of a typical case are characteristic there are very many cases which are atypical. The sudden onset with severe chill, the rusty sputum, the ordinary temperature curve, the suggestive herpetic eruption upon the face, and the disturbed pulse-respiration rate may all be absent, requiring us to depend upon the physical signs indicative of rapidly progressive lobar consolidation. The variations in type as observed in different classes of individuals are misleading, also cases with a very limited lesion but grave general phenomena. The prominence of nervous symptoms in children and drunkards, the frequent absence in the aged of a high temperature range, as well as of cough and expectoration, also the frequent absence of symptoms referring to the lungs, and the presence of symptoms of a typhoid character, are some of the conditions leading to obscurity in diagnosis. From acute phthisis of the pneumonic type a separation, without the help of the microscope, is often impossible.

Pneumonic fever is differentiated from catarrhal pneumonia with ease in most cases. Catarrhal pneumonia is not as rapid in onset, usually supervening upon some primary affection, such as simple bronchitis, whooping cough, measles, etc. It is usually attended by more cough, the sputum is not rusty, and largely on account of the involvement of the finer tubes, dyspnoea is more marked than in pneumonic fever, the respirations being often exceedingly rapid and the surface cyanotic. The physical signs generally indicate a bilateral consolidation, the areas of consolidation being smaller and the upper lobes more likely involved



than in pneumonic fever. There is an addition of the râles of bronchitis, both large and small; fine, closely aggregated moist râles being often heard over the consolidated areas. It is seldom that there is a well-developed bronchial or tubular breathing, but this sign may develop slowly from a coalescence of consolidated and collapsed areas. If attended by an uneventful onset and a development of typhoid symptoms, pneumonic fever sometimes presents a marked resemblance to typhoid fever, for which it is sometimes mistaken, this error being the more readily made on account of the occurrence of a pneumonic complication in an occasional case of typhoid fever, even in the early stage, and frequently, of a variety of symptoms and signs calling attention to the lungs. Much depends upon the relationship to each other, in point of time of development, of the signs of lung consolidation, and the typhoid state.

The frequent occurrence of pneumonic fever late in the course of a variety of chronic diseases, notably diabetes, Bright's disease, etc., often without a prominence of characteristic symptoms, makes it easy to pass it by without detection. In young children the chest should be investigated daily if a group of symptoms is present which makes it at all suggestive that pneumonia may exist. Cerebral symptoms, abdominal pain and diarrhœa, high fever, without marked symptoms calling attention to any organ, may each be attendant upon pneumonia in young children, with but the slightest cough, etc. I have repeatedly seen in children pleurisy with liquid effusion mistaken for pneumonia—and at times, without the use of the aspirator, this is a pardonable error, often due to the presence of the vocal signs of consolidation and bronchial breathing. But in general the error is due to inefficiency in physical exploration.

Strümpell lays stress upon the importance of examining the lateral portions of the chest and the axillary regions, as râles are often heard first in these locations, in pneumonia of the lower lobes. He also calls attention to the necessity of examining the posterior portion of the upper lobe, as in apical pneumonia the consolidation is often first discoverable there; also to the first signs of infiltration being often first detectable posteriorly in the middle of the thorax, extending downward from that point.

**Prognosis.**—Fox places pneumonic fever third in the list of fatal diseases. Under good treatment, and even with good nursing, the majority of cases of pneumonic fever occurring in healthy individuals, not in the extremes of life, recover fully. But in the old, the debauched, those suffering from chronic maladies, especially diabetes, Bright's disease, heart disease, etc., the mortality is very high. The percentage of deaths in persons over sixty years of age was given by Grisolle at 59 per cent. Statistics show a steadily increasing mortality with each decade of life beginning with the twentieth year. The general mortality, in un-

selected cases, as they are received into the hospitals of this country, varies from 12 to more than 40 per cent. The mortality varies much in different years, and also in different regions, being greater in our southern states than in the northern.

Complications exercise an important influence. Of these, purulent meningitis is perhaps the most fatal. The various cardiac inflammations often determine a fatal issue, especially endocarditis. Failure of the heart is indicated by evidences of overdistension of the right ventricle, pulmonary œdema, cyanosis, etc. Failure may occur early without these symptoms of venous stasis, and appears due to the action of the specific poison upon the nervous apparatus of the organ. The temperature possesses a decided significance. A temperature under 104° F. is favorable. A continued rise of temperature after the fourth day is unfavorable. A low temperature range is encouraging if the respirations are not much accelerated and the general symptoms correspond. A rapid pulse (above 120) and disturbance of rhythm, or intermittency, are of bad omen. Offensive expectoration suggests gangrene, and the prune-juice fluid is evidence of advanced deterioration of the blood and tissues. Loud tracheal râles usually suggest the approach of the end and are generally, not always, associated with inability to expectorate. In this stage pulmonary œdema is frequently present.

Death is rarely the result of asphyxia, due to the extent of the inflammation; much oftener of failing circulation, or the general influence of the poison. Complications determine many of the fatal issues.

The rapid development of the physical signs of consolidation, a large quantity of frothy expectoration, a respiration rate of 50 or more, a rise of temperature, and an increased frequency of pulse, from the seventh to the ninth day, continued delirium during the twenty-four hours, are all of ill omen.

In an analysis of 10,000 cases of pneumonia treated in the London Hospital, Fenwick found that the amount of albumin in the urine possesses decided prognostic value. The mortality was shown to be directly related to the height of the fever, and cases beginning with severe gastro-intestinal symptoms presented double the mortality of those in which a chill initiated the attack.

The influence of leucocytosis upon the prognosis of pneumonia is important. It has been recently studied afresh by Cabot and by Carini, their conclusions indicating that while the presence of leucocytosis cannot be considered as a very favorable sign, its absence warrants an unfavorable opinion.

It is quite an old observation that the white blood corpuscles in the blood are increased during an attack of pneumonia, but Kikodze has recently shown that such a leucocytosis is not present in cases which end fatally. Schestowitsch inoculated rabbits with the pneumococcus

and observed that only those developing leucocytosis lived, while in the fatal cases the white corpuscles were diminished. Von Takish has made the same observation, and has recommended that only drugs be used in the treatment of this disease which produce an increase in the white corpuscles.

**Treatment.**—The great prevalence of pneumonia, and the large number of persons who annually die from it, justify a thorough consideration of its therapeutics. In determining the value of any treatment of pneumonia, its clinical history must be well understood. When we recall that it is an acute disease with an inherent tendency to terminate spontaneously by crisis, any time between the second and the ninth to the twelfth days, and add to this the varying character of different epidemics, differing as much as do epidemics of diphtheria or typhoid fever, the effect upon its course of age, constitution, and perhaps of unknown epidemic influences, the difficulty attending the comparison of epidemics, as well as of individual cases, in order to arrive at the comparative value of treatment will be fully appreciated.

The treatment of pneumonia upon homœopathic principles is in the main quite satisfactory. I think we may as homœopaths congratulate ourselves not only upon the small mortality among our patients, but also upon the modifying influence our method has exercised upon the practices of the dominant school. In support of these statements I have but to quote Dr. Wilson Fox, who in his article upon *Pneumonia* in Reynolds' "System of Medicine," in speaking of venesection, says: "The treatment thus indicated continued in use, with more or less freedom, in this country until attention was forcibly drawn by Dr. Balfour to the lesser mortality of pneumonia in Skoda's practice, and also in some of the homœopathic hospitals where bleeding had for some time been discontinued." This paragraph refers especially to the Vienna homœopathic hospital in charge of Drs. Caspar and Wurmb. As stated by Dr. Fox, bleeding had been discarded by these physicians, who reported a mortality of only 6 per cent. against a mortality of 18 per cent. occurring in the other Viennese hospitals, and it seems in every way reasonable to suppose that such a convincing demonstration led Skoda, the distinguished Vienna diagnostician, to his avoidance of the lancet.

**GENERAL CARE OF THE PATIENT.** As usual in the care of persons suffering from infectious disease, the hospital apartment should be roomy, well ventilated, and preserved at a temperature of from 68° to 70° F. It is often an advantage to develop a humid atmosphere, which can be accomplished by means of an atomizer or a large pan of water upon a stove. From the beginning of the attack the patient should be kept thoroughly at rest, in recumbency, upon a firm mattress. He must not be allowed to assume the sitting position for purposes of physical examination of the chest, changes in clothing, or for any purpose what-



ever. Company should be interdicted. The position which confers the greatest amount of comfort should be permitted; this is generally upon the affected side. The diet should be simple, easy of digestion, and generally exclusively liquid—milk, koumiss, junket, whey, animal broths, gelatin, eggs shaken with milk, to which in suitable cases may be added an alcoholic stimulant, are some of the most suitable articles. It is wise to invariably prescribe precise quantities of food to be administered, as well as the times of administration. Overfeeding is to be avoided, especially if symptoms of gastric, or gastro-intestinal irritation exist. It is important that the patient take plenty of water, which may be flavored, if desired, with lemon-juice, tamarinds, the juice of raspberries, apples; also a little brandy or porter added to the water is often appreciated. The patient should be kept clean by means of soap and water, a process which is without danger of resulting cold or prostration, if properly conducted.

The bowels should be emptied by means of enemas, and in some cases it is best to give at the outset a mild laxative, if the bowels have been torpid and the secretory organs inactive. The bladder should be watched, as in low forms of pneumonia this organ is often distended, demanding the catheter.

LOCAL APPLICATIONS. Poultices and hot fomentations to the painful side are sometimes advisable. When pain is great, many, following the practice of the Germans, apply ice-bags to the affected side. Others, especially the English, recommend loosely quilted cotton jackets and protect the patient with the utmost care from the slightest of chilling applications. As each claims good results, one must conclude that neither are specially valuable. My own experience corroborates this view. In general, protection of the chest with one layer of flannel and a cotton night dress is sufficient.

STIMULANTS. In cases in which the digestive power seems good, the heart not flagging, and the fever running a normal course, alcoholic stimulants are unnecessary; the reverse suggesting their use. Feebler digestive power, if benefited by alcoholics, will improve under small quantities administered with the food. High temperature is as a rule but slightly affected, even by large quantities. The influence of alcohol upon the failing heart is very valuable; sometimes very small quantities, *i. e.*, teaspoonful doses every hour or two, will produce a very appreciable effect. In others, a half ounce to an ounce is required to secure the same result, and in quite a percentage of cases there is no appreciable influence from the alcoholic. In general the stronger alcoholics are preferable, *viz.*, whiskey or brandy; when a mild stimulant is required, wine whey is to be recommended. Porter, water, or a little lager beer is often enjoyed. In serious degrees of heart-failure a diffusible stimulant such as champagne may be given with excellent results, if given in large quantities.



In grave cases I have given from a pint to a quart within a period of four to six hours with the happiest results. Of one thing I am satisfied, that in order to secure the beneficial action of alcoholics in high grades of heart-failure, they must often be given in quantities which appear to be enormous. Under such circumstances I have rarely observed the slightest symptoms of alcoholism.

**MEDICINES.** In the early stage (hyperæmia) there are four medicines which possess strong claims for attention, and usually there is little difficulty in differentiating them. I refer to aconite, bryonia, ferrum phosphoricum, and veratum viride. *Aconite* is highly esteemed by physicians who do not find it necessary to administer it in large doses. Those who do, soon abandon it, on account of its depressing influence upon the heart. It is pre-eminently indicated for sthenic cases, presenting a hard pulse and a rapid rise in temperature following a sharp chill. The patient is usually nervous, restless, apprehensive, the skin hot and dry, and a marked thirst. Prescribed in the lower dilutions for these cases, aconite frequently modifies in a very decided manner the entire course of the disease. *Ferrum phosphoricum* is preferable for feebler individuals whose systems do not react so sharply against the poison, especially those who are suffering from debilitating chronic disease, or some acute affection, such as measles; or in feeble, anæmic persons. The chill is not as pronounced, the temperature does not rise as rapidly, there is less of nervous excitement, than in the aconite patient. There is quietness, often drowsiness, and the expectoration quickly becomes rusty, or contains much blood. This remedy is especially useful for secondary pneumonias associated with pulmonary phthisis, measles, or any of the infectious diseases. Marked pleurisy contraindicates it, while the existence of bronchial symptoms favors the selection of this medicine. I have been fortunate enough to see two cases of pneumonia, in the first stage, presenting the ordinary symptoms and the typical crepitant râles diffused over the lower lobe of the lung, abort under the action of ferrum phosphoricum in the second decimal. No remedy in the *Materia Medica* has a stronger claim to a position among the anti-pneumonic remedies than *bryonia*, judging from both its provings and from clinical experience. Its action upon the lung parenchyma, pleura, and bronchial tubes, has been thoroughly established by means of poisoning cases and provings. It is without question our most important medicine for pneumonia associated with pleurisy, and should be first administered unless satisfactory indications for some other medicine exist. The patient is usually found resting upon the affected side and complaining of stitching pains, dyspnœa, a dry mouth, and a good deal of thirst. This remedy is usually promptly effective in relieving the pleuritic pain, and under such circumstances favorably modifies the future course of the disease.

*Veratum viride* is very popular in our western states for the early

stage of pneumonia when arterial excitement is great or the lung becomes rapidly gorged with blood. Hale recommends one or two drops of the tincture repeated every half hour in urgent cases. Veratrum viride, like aconite, is a powerful cardiac depressant, and when used in the tincture should be prescribed in doses which will fall short of producing an unpleasant effect.

Of the quartette of medicines recommended for the first stage of pneumonia, two are equally valuable for the stage of consolidation, viz., ferrum phosphoricum and bryonia. These are medicines which may be continued through the second stage, if indicated. It is the practice of some to continue aconite or veratrum viride in association with whatever medicine is decided upon as most suitable for the stage of full development, providing the medicine has made a favorable impression upon the disease in the first stage.

*Phosphorus* undoubtedly possesses the greatest reputation of any medicine for the treatment of pneumonia after the early hyperæmia has subsided. It owes its early reputation to Fleischmann, of Vienna, one of the early homœopathists, a man fond of treating large groups of cases with a single remedy. Applying phosphorus in this manner to the treatment of pneumonia, he was able to report 377 cases with a mortality of 19, or 5 per cent. The last 78 all recovered. The mortality in the general hospitals at the time was 19 per cent. and upwards. My own experience with phosphorus has suggested its use in pneumonias in which the element of hyperæmia is more prominent than the exudative feature. Pneumonias of this type are common, being especially frequent in association with influenza. It appears to be well suited to delicate persons in middle or advanced life. The presence of pleurisy does not contraindicate it; indeed I have been rather impressed by its influence upon this feature of the disease during our recent epidemics of influenza. The development of typhoid symptoms—the so-called typhoid pneumonia—suggests a consideration of phosphorus. The most satisfactory dose is one drop of the second decimal dilution repeated every two to four hours. Old preparations of phosphorus are unsatisfactory.

*Sulphur* has been generally recommended as a remedy for the third stage of pneumonia. The author has in many cases followed the advice of the late Dr. Hering to give it in the early stage of uncomplicated pneumonias not calling emphatically for another medicine. His advice was to administer sulphur as soon as the nature of the case was apparent, accompanying or following sulphur by any medicine apparently indicated, such as aconite or bryonia. Pathologically it appears to be better suited to cases with rapid development of pulmonary consolidation, and in this respect is unlike phosphorus. Sulphur is very successful in pneumonias of the old, also in the pneumonias of children with cerebral symptoms simulating meningitis or actual meningitis, viz., rolling of the head,

squinting, stupor or complete unconsciousness; or the child may show great irritability, have frequent flushes and red lips. A forenoon aggravation strengthens the indications. The third decimal trituration repeated every two hours is a satisfactory method of administration.

*Iodine* and the *iodides* are remedies infrequently used by the great majority of physicians, and yet unknown riches exist in the group. Recent clinical experience with iodine à la Fleischmann, indicates its position in the front rank of medicines for pneumonia. Allopathic observers have administered it in drop doses of the tincture every few hours, but at least in many cases one to two drops of the first decimal dilution is sufficient.

The *iodide of antimony* is an efficient remedy for pneumonia associated with a marked bronchitis, a frequent feature of pneumonia as it appears in phthisical subjects. The expectoration is muco-purulent, perhaps bloody, or may present the rusty fibrinous character of pneumonic sputum combined with the former. Experiences with the iodide of antimony to this date suggest it as a remedy of great power in pneumonia, and I would commend it to the profession for careful investigation.

The *iodide of tin* is a promising remedy for the stage of purulent infiltration. It has proven beneficial for the condition represented by loud moist râles in the larger bronchial tubes, localized moist râles over a portion of the consolidated lung, expectoration with difficulty, of quantities of heavy yellowish-brownish matter with some odor—a group of symptoms suggestive of a destructive lesion. The second decimal trituration only has been employed. It should be remembered that all iodides deteriorate with age.

*Tartar emetic*, the medicine so long abused and now seldom mentioned by the dominant school except to condemn, is, when properly administered to suitable cases, the peer of any known medicine in the treatment of complicated pneumonic fever. The old school have endeavored to see how much, and the new school how little, could be administered, but the best plan appears to be one applicable to many medicines, viz., to prescribe such doses as to keep just short of developing its physiological action. This method has proven promptly successful in a number of cases in which smaller doses had failed. Numbers of my colleagues can bear testimony to this statement. For adults two grains, and for children one grain of the second decimal trituration repeated every two or three hours appears to be the most suitable dosage to begin with. The method of administration is of such vital importance to success in the use of this medicine that I have allowed these remarks to precede a consideration of the indications for its use. This medicine is not indicated in sthenic pneumonia, the error of those who first used it being the endeavor to break down cases of this character by its use. It is rather the asthenic pneumonias of the young and the old which call for its use.



The most important symptomatic indications are the well-known symptoms growing out of pulmonary failure, viz.: loud râles in the larger bronchial tubes, or a multitude of subcrepitant râles indicative of pulmonary œdema, cyanosis and a cool, moist skin.

A rival to this medicine has recently appeared in the character of *arseniate of antimony*, a medicine which has proven useful in the pneumonias of elderly people, and especially such as have complicated *la grippe*. The writer has observed two cases, in association with physicians of this city, in which the group of symptoms designated "paralysis of the lungs" have been present, promptly improved under the use of this medicine in the second decimal trituration, the symptoms being severe dyspnœa with loud rattling in the bronchial tubes; frothy watery sputa expectorated with difficulty, followed by inability to evacuate the tubes; a feeble, rapid (and in one case intermittent) pulse, and all the surface indications of failing circulation. This remedy is well suited to pneumonia occurring in persons with some underlying organic disease of the heart or kidneys.

The group of pneumonias in which the typhoid state is present, frequently call for another class of medicines, the most important members of which are hyoscyamus, rhus toxicodendron, agaricus, and less frequently, arsenic, baptisia and phosphoric acid. Hyoscyamus easily holds the first position in the treatment of cases with cerebral symptoms, the various forms of mild and grave delirium being most frequently controlled by this remedy. Its action has been apparent even when the patient has lain in a stupor with perhaps incoherent muttering. It often controls the distressing cough and the profuse sweatings occasionally met. The most violent symptoms of cerebral irritation may be present, with limited consolidation of the lung. In this class of cases the poison expends itself upon the system at large rather than upon the lung.

If hyoscyamus fails to control the cerebral phenomena, agaricus or its active principle agaricine may be considered. This remedy is sufficient for the highest grade of cerebral symptoms. The patient is sleepless, restless, looks pale, anxious and makes attempts to escape. Profuse sweatings and a feeble heart strengthen the indications for its use. It is valuable in the pneumonias of drunkards. If the tincture is given, ten drops may be added to four ounces of water, of which teaspoonful doses may be repeated according to the urgency of the symptoms; or of agaricine, which is preferable if the heart is very feeble, two grains of the first decimal trituration should be given hourly.

The other members of this group are prescribed on their well-known indications, many of which have been detailed in the article on typhoid fever. I would call attention especially to the value of phosphoric acid in the quiet apathetic form met most frequently in elderly persons with mental dulness but no delirium. A successful method of administration has been the addition of ten drops of the pure acid to four ounces of water, of which teaspoonful doses should be given hourly.



*Circulatory failure* is the most frequent cause of death in pneumonia. It is due to various causes, viz., the action of a poison, overdistension of the right heart, high temperature, etc. To prevent this condition we must control these causative factors. The influence of heat upon the heart in pneumonia is probably overestimated and antipyretic measures employed too frequently; cold spongings or the cool bath are most to be recommended. Of late the "ice-cradle" has been popular for the same purpose. The medicinal antipyretics, such as phenacetin, acetanilid, are considered by most observers as objectionable, although doses of three grains of either of these drugs will usually produce a prompt reduction in temperature; nearly all of the bad results, however, have arisen from the administration of large doses (five to ten grains). The possession of remedies quite certain in their action upon the temperature presents a temptation to their abuse. The influence of the toxic agent upon the heart can only be positively modified by medicines. The same must be stated of the engorged heart. If the heart enfeeblement progresses in spite of the best-directed efforts it becomes necessary to make use of medicines for their stimulating influence upon the organ. Our first recourse should be to alcoholics, the use of which has been discussed. An agent used much of late for the control of this and other symptoms of pneumonia is oxygen; some make use of the pure oxygen gas, while others employ it mixed with 20 per cent. of nitrogen monoxide. To be of benefit oxygen must be used freely; the frequency of the inhalations being governed by the effect. In favorable cases its beneficial action is indicated by a lessening in dyspnoea, cyanosis, and in the frequency of the pulse, which becomes a little fuller and perhaps a little more regular; from one to five gallons may be given in an hour, in urgent cases. Great care should be exercised in its administration to see that the oxygen is really inhaled, not lost. Failures due to the latter cause have been observed. Of the medicines available for this symptom I have, during the past year or two, used agaricine with considerable satisfaction. To the importance of this medicine as a cardiac stimulant I have repeatedly called attention in the medical journals. The dose employed has varied from one to three grains every one or two hours. The sulphate of sparteine in similar doses is less frequently effective. Nitro-glycerin in a 1 per cent. solution, repeated every half to two or three hours, is sometimes effective. The remedy which at present possesses the greatest reputation for the control of this symptom is strychn. sulph., which is administered in tablets of one hundredth of a grain every three to six hours, or in urgent cases it is better given hypodermatically in doses of one fiftieth of a grain.

Venesection, "the lost art," is again coming into favor as a means of treating strong plethoric individuals during the congestive period, or later, when great frequency of respiration with frothy expectoration and

loud rattling in the chest suggest collateral œdema of the lung area not involved in the pneumonic process. Cases of this character involve great danger to life, and if unrelieved, this measure should be considered.

Interesting experiments by the Klemperer Brothers, into the question of immunity from this disease, resulted in the discovery that every nutrient medium in which the pneumococcus is inoculated, will render rabbits immune against pneumonic septicæmia, and this after the cocci have been removed by filtration. The duration of the period of immunity secured, was seldom more than six months. Offspring born within this period were also immune. The same observers have been able to secure immunity in other animals by means of the introduction of serum taken from those already immune, and still more important, to promptly control by this means the artificially produced pneumonic septicæmia. In the latter experiments the serum was injected twenty-four hours after infection, the animal having at that time a temperature varying from 105° to 106.5° F. The quantity of serum used was two drachms, with the result of a gradual depression of temperature during the next twenty-four hours. In twelve consecutive experiments a successful issue was obtained. These experiments have been successfully repeated by other investigators.

The theory advanced to explain these results is as follows: The pneumococcus elaborates a poisonous albumin called pneumotoxin, which in the blood of an animal induces the development of a secondary albuminous product which possesses the power of neutralizing the influence of the primary poison. What is thus experimentally induced in animals is claimed to occur in man, *i. e.*, the blood developing a substance antidotal to the poison given to it through the action of the specific germ in the lungs. The crisis occurs when the antitoxic element overcomes the toxic. This antitoxic agent they have demonstrated to exist in the blood serum of persons who have passed the crisis of pneumonic fever, and in a number of instances its injection has cured the experimental disease in animals. In the human species the same experiment has resulted favorably in a number of cases.

The same observers have also discovered that this serum not only grants immunity from pneumonic fever but will quickly cure the disease in the form produced in animals by inoculations, even if introduced after the disease has become established.

Audéoud prefers to make the injections into the subcutaneous cellular tissue of the thigh, and advises that the syringe be carefully warmed and sterilized and the skin thoroughly cleansed. Transfusion of blood from persons convalescent from pneumonia has been practised by Hughes and Carter. In twenty-five of thirty cases treated, the crisis seemed to be precipitated.

Mays has of late strongly advocated the application of ice to the

chest in croupous pneumonia. He advocates thoroughness in the treatment, *i. e.*, applying to the front, back and side of the chest rubber ice-bags. He also applies ice to the head. The bag should be well wrapped in flannel and be kept continuously applied.

The treatment of pneumonia by means of cardiac stimulants has been extensively practised during the past few years, and extraordinary results claimed. The drugs used have been especially digitalis (which has been administered in enormous doses by Petrescu), strychnia, strophanthus, etc.



## FEBRICULA.

**Synonyms.**—Simple continued fever; ephemeral fever.

**Definition.**—The term febricula includes cases of essential fever, not due to any definite lesion or known specific infection, and not associated with changes in any of the organs of the body. It is spoken of as ephemeral fever when lasting but two or three days.

**Etiology and Pathology.**—There is now a general feeling that febricula is very often but the symptomatic expression of mild inflammation or abortive cases of infectious disease. During epidemics of typhoid fever, scarlatina and measles, cases susceptible of this explanation are often seen. The possibility of overlooking the existence of small inflammatory foci in different portions of the body is perfectly conceivable. Cases also occur apparently as the result of indigestion; some are due to the taking of decomposed food and drink. Those occurring after exposure to the sun are believed to be examples of mild thermic fever. Aside from the cases occurring under the circumstances above detailed, there are numerous others in which none of these causes are discernible, and to which the name “simple continued fever” strictly applies. These appear most frequently in children and adolescents, possessed of a febrile predisposition. They are often excited by slight mental and bodily fatigue.

**Symptoms.**—The onset of simple continued fever is usually abrupt. Exceptionally, prodromic symptoms, a chill, or in highly neurotic children, a convulsion, may be observed. The temperature is rarely high, ranging usually from 101° to 103° F. The associated symptoms are the usual concomitants of pyrexia in nervous subjects, viz., restlessness, mild delirium, and muscular twitchings. The fever lasts from three to ten or twelve days, when defervescence takes place by crisis, which is often accompanied by critical discharges, as sweat, epistaxis or diarrhoea. Herpes about the mouth is very common.

**Diagnosis.**—The recognition of simple continued fever, it will be seen from the above description, rests solely upon negative evidence. The sudden rise in temperature at once suggests the possibility of *scarlatina*, from which it is soon distinguishable by the failure of sore throat or the characteristic rash to develop.

The greatest difficulty is in differentiating it from *typhoid fever*. This is done by the absence of enlarged spleen, abdominal rash and Ehrlich's urinary reaction.

Cases occurring in malarial districts may be confounded with *malarial*

*fevers.* In the latter we have enlarged spleen and a characteristic temperature curve.

**Prognosis.**—This is uniformly favorable. Cases occurring in very young or old, debilitated subjects sometimes present cause for alarm. Proper treatment generally puts an end to the disease within a few days.

**Treatment.**—Owing to the possibility that there may be hidden changes not discoverable by our present methods of physical exploration, cases must be treated with all the care bestowed upon more dangerous diseases until the diagnosis is assured. The patient should therefore be put to bed and kept at absolute rest. The diet had better be restricted to milk and other light foods. Sponging of the surface of the body with cool water is very grateful.

The principal remedy for febricula is *aconite*. *Belladonna* is often indicated in children. It may be administered in from 1x to the 3x dilution, at intervals of from one to three hours, according to the demands of the case. Other remedies useful when *aconite* fails are *gelsemium*, *baptisia*, *rhys tox.* and *arsenicum*.

## ASIATIC CHOLERA.

**Definition.**—Cholera is an acute specific infectious disease, caused by the “comma bacillus” and characterized by violent purging (the stools being of a serous character), vomiting, cramps, and collapse.

**Synonyms.**—Cholera; cholera algida; epidemic cholera; cholera maligna, etc.

Asiatic cholera finds its home in India, in which country, and particularly in that portion of it known as Hindostan, only is it endemic. There it has been known since ancient times. It has only been in the nineteenth century, with its greatly increased facilities for travel and intercourse between the nations of the world, that the disease has extended to Europe and North America. The earliest *well-recorded* epidemic of an extended character in India is that of 1817. A second great epidemic occurred in 1827. This extended to Russia by the year 1829, reaching Great Britain and Germany in 1831, and North America in 1832. This was followed by lesser epidemic developments, which gradually abated until 1839, when the disease disappeared, comparatively speaking. Since that date epidemics have occasionally occurred in North America. In 1848 one gained a foothold at New Orleans, and thence spread northward through the Mississippi Valley. In 1849 and again in 1854 epidemics appeared, both times commencing in New York City. Slight epidemics appeared in 1866 and 1867, and finally in 1873, since which time, thanks to careful quarantine regulations, cholera has at no time gained an entrance into the United States, notwithstanding the danger incurred, especially in 1892, from the entrance of infected vessels from abroad into New York waters. The Russian epidemic of the latter year was one of exceptional fatality, official reports indicating 265,760 deaths from cholera. Such a high mortality was due very largely to the epidemic following closely upon the heels of a great famine in the infected country.

**Etiology.**—The announcement by Koch in 1884 that he had discovered the specific cause of cholera Asiatica in the form of a peculiar spirochæte, was welcomed on every hand. The observations leading to this discovery were made by him in India during 1883 and 1884, while acting as chief of the commission sent out by the German government, for the purpose of studying the etiology of cholera. His observations have since been abundantly confirmed by numerous observers, who have shown that the “comma bacillus,” as the cholera bacillus is commonly called (because of its peculiar curved conformation), is constantly

present in this and no other disease. Its habitat is the small intestines. Cholera bacilli present varying degrees of curvature; ordinarily consisting of a simple curve, as that of a comma, they may at times resemble more closely the letter S. The length of the spirillum is 0.5 to 1.0  $\mu$ . The typical Koch's vibrio has a single flagellum at the free end. Such variations in size and appearance are due mainly to the fact that two or more bacilli are often attached to each other at their extremities. It develops by fission. Spores have not yet been demonstrated. They exist in the stools of the earliest stages of the disease, and may be found in the rice-water dejecta in great numbers; the vomited matters seldom ever contain them. The growth of these micro-organisms outside of the body is characteristic. If kept moist or in water their vitality may be retained for a long period of time. They are, however, readily destroyed by dessication; acid substances also destroy or retard their development, which accounts for the growth of the germ in the intestines and not in the stomach. Growth is most active at 30° to 40° C. and ceases at 16° C. Still they do not die at even a much lower temperature, even at 10° C. They are destroyed, however, at a temperature of 60° C. Their growth is easily established upon agar, gelatin, beef tea, potatoes, etc. They are actively mobile in fluids. Rietsch, Nicati, and Koch, have developed choleraic symptoms in guinea-pigs by the introduction of the cholera bacillus into the duodenum.

Animals which have been supposed to be immune to cholera, have been shown by Pasteur to develop the disease following experimental inoculation. Guinea-pigs and dogs were employed.

The development of immunity in animals, by means of attenuated cultures, has been claimed by Gamaleia and Lowenthal.

The morphological characteristics are not alone sufficient for the identification of the cholera bacillus. Cultivation experiments are necessary. The germ has but a short life-tenure, and in order to preserve the species it is necessary to make frequent cultures. The organism is found in the early stools, frequently disappearing by the fourth or fifth day, and is seldom present after the eighth or tenth day. Morphologically related bacilli have been found in the apparently normal secretions of the mucous tracts, and also associated with various disorders of the bowels, such as dysentery, and various diarrhœic affections.

During the early years of our knowledge of the "comma bacillus," it was supposed that this organism possessed characteristics clearly separating it from other species; but further observations have demonstrated that the vibrios which have been obtained from different epidemics are not identical, and it seems probable that there is a large group of germs presenting similar characteristics, many of which have no relationship to cholera. As already indicated, efforts to differentiate by means of the microscope have not been satisfactory, on account of the varying morpho-



logical characters presented at different periods of growth of a single variety. The only recourse under these circumstances is to the information afforded by cultivation methods. Max Gruber in his excellent *Lancet* article (July 7, 1894) says, "further investigations have, however, convinced me that all kinds of cholera vibrios present characteristic appearances in gelatin plate cultivation, and I would venture to distinguish by means of gelatin plates the Koch's vibrio from all similar species with the exception of Deneke's vibrio." The evidence at present in our possession indicates that the cholera bacillus does not penetrate the organism in numbers, but develops in the intestines, the poisoning of the individual with the resulting symptoms being through the agency of a tox-albumin generated by the bacillus.

The spread of cholera seems to be chiefly, if not entirely, due to carelessness in disposing of patients' dejecta, which contain the specific organism; many of these stools represent a nearly pure culture of the bacillus. The propagation of the germs outside of the body goes on freely upon moist clothing or surfaces of varying character to which they may have become attached, in impure water, upon moist soil, in milk, and upon the surfaces of fruits and vegetables. The manner in which they gain entrance to the interior of the body can now be readily imagined. Undoubtedly contaminated water is the principal, some say almost the only, medium. This explains, in connection with subsequent statements, why nurses, laundresses, and scrub-women are attacked more frequently than others about the sick. The slow spread of the disease along the international highways, evidently not more rapidly than intercommunication of individuals can account for, indicates that the disease is not disseminated by the atmosphere, but rather that it is due to an element which must be carried to the individual in or upon some substance. The influence of a contaminated water supply in developing and continuing an epidemic is most important. A direct relationship may be stated to exist between the degree of impurity of the drinking water and the virulence of an epidemic. This has been repeatedly verified, and never more clearly than during the terrible Hamburg epidemic of 1892. Koch thinks that the bacillus develops especially well in briny water. That the cholera spirillum finds entrance by way of the digestive tract is indicated by the presence of the germ in the intestines only. The clinical fact that gastric catarrh predisposes to the development of cholera suggests that in healthy conditions of the stomach the cholera germ is destroyed in transit to the intestines.

Epidemics of cholera usually occur in the summer. They develop in the East early in the season, and reach American shores some time between mid-summer and frosty weather.

Liability to the disease is general. Children are not, however, so frequently attacked as adults. Sex exercises no influence. Old people

are especially susceptible. Most important in the etiological relations of cholera is the condition of the gastro-intestinal tract, catarrhs and disorders of which are powerful predisposing factors.

Cholera has appeared in all regions except the extreme North; cold, therefore, is an opponent to its extension, which is also demonstrated by the frequent instances of disappearance of an epidemic during the winter months and its reappearance with the warm weather of spring.

Such neglect of personal hygiene and general sanitation as occurs among the poor living in crowded quarters is in the highest degree favorable to the development and spread of cholera. The spread of the disease is inhibited by separation of the inhabitants, the practice of greater personal and local cleanliness, and the supply of larger amounts of pure water.

Predisposing causes are especially periods of want, famine, debility from any cause, abuse of alcohol, overwork, etc.

**Morbid Anatomy.**—Cholera does not possess a specific lesion. The stomach at most shows what appears to be a mere catarrh. The most important anatomical changes are found in the small intestines. These consist of the alterations peculiar to catarrh of that locality. With full development of the intestinal process, the solitary glands and Peyer's patches become swollen and reddened. The mucous membrane may even become the seat of numerous ecchymoses. Desquamation of the epithelial cells is very extensive. According to some authorities this latter explains the profuse serous exudation into the intestines. Others, however, believe the desquamation to be a purely post-mortem alteration. The changes at a later stage are often of a pronounced diphtheritic character, the ulcerated and necrosed intestine yielding a sanious offensive fluid in marked contrast to the earlier clear rice-water discharges.

The rapidly fatal issue which occurs so frequently, leads to an early and a most pronounced *rigor mortis*, post-mortem distortions of the eyes and movements of the jaws and limbs being common. As might be expected when one considers the great serous drain, the tissues are pale and dry, *i. e.*, bloodless. The left ventricle is contracted. The lungs are collapsed. They may be hyperæmic, especially in the lower lobes. The blood, contrary to the general rule in infectious diseases, occupies mainly the venous system. It is, moreover, thick and dark from great loss of its water and salts. The spleen is not enlarged. The kidneys show cortical congestion, the microscope demonstrating parenchymatous nephritis. In cases passing quickly to a fatal termination, there is an absence of a marked degree of inflammatory changes, the entire body being pale and shrunken.

**Clinical Course.**—The active symptoms of cholera develop after a period of incubation ranging in duration from one to four or five days. The intensity of the attack itself may vary within the widest limits.

Many cases are so mild that they could not be identified as cholera, were it not for the prevalence of an epidemic of that disease. On the other hand there frequently occur cases of such marked severity as to end fatally within a few hours. Naturally these variations in intensity of the disease lead to different clinical pictures presented by different cases. Thus has originated a number of terms applied to cholera in its various phases. The light cases, if passing quickly to recovery, are designated "choleraic diarrhœa." If, however, the diarrhœa continues for a few days and then merges into a fully developed cholera, the term "premonitory diarrhœa" is applied to it. The symptoms of a light attack are those of an intestinal catarrh. The stools are profuse, watery, usually painless, and may be repeated from every two to six or eight hours. There are some prostration, loss of appetite, and perhaps a spectre of true cholera appears in the shape of vomiting, slight cramps in the calves, diminished secretion of urine, etc. Still another term, "cholerine," is made use of to designate a variety intermediate between the mild type above described and true cholera. It resembles closely *cholera morbus* as we see it here during the autumn and late summer months. The attack is sudden in onset; diarrhœa is soon followed by vomiting, the surface of the body becomes cool, the voice weak, the pulse feeble; cramps appear in the calves of the legs; the urine may be scanty and show a little albumin. These symptoms may continue with varying degree of severity for one or two weeks.

Fully developed cholera often begins with a slight premonitory diarrhœa, which may or may not be preceded by a day or two of colicky pains and other symptoms of prodromal character. In other instances the symptoms of the disease appear promptly without the slightest warning whatever. These consist of an increasing diarrhœa, to which are soon added vomiting, retention of urine, cramps, and all the phenomena of collapse. This is the "algid stage," or stage of collapse. It lasts for one or two days. The stools now become rapidly more frequent, and soon consist of copious "rice-water" dejecta, almost without color or odor. After standing they deposit a whitish granular sediment, consisting of epithelial cells, triple phosphate and a variety of micro-organisms. Blood is occasionally present. Most important among the bacteria present is the cholera bacillus. These can be readily detected with the microscope, but pure cultures must be made before one can positively identify them. Abdominal pain and tenesmus may be present.

The evacuations, in rare instances, may be infrequent, or even absent, and this, too, in severe cases. This type has been called "*cholera sicca*." Under these circumstances the intestines have been found full of fluid, the failure to have an evacuation being probably due to a paralysis of the muscular coat of the bowels.



Vomiting is not delayed long after the diarrhœa has appeared. It may be very frequent and distressing; but this is not the rule. The vomited matters at first represent the fluids taken, but later consist of a fluid which resembles the stools, and which is a transudation through the mucous membrane of the stomach and upper portion of the small intestines. The tongue is coated, the appetite gone, and thirst usually intense. Hiccough may attend the vomiting. The abdomen is flat or concave. Sometimes fluctuation, due to accumulation of fluid within the intestines, may be detected in the abdomen.

Succeeding the diarrhœa and vomiting, and adding to the symptomatology of cholera, is a most characteristic group of phenomena, that of failure in the circulation. The surface of the body becomes cool and shrivelled and covered with a clammy perspiration. The features are sunken and the color ashen. The surface temperature is depressed, but the rectum may show 102° F. The pulse becomes feeble, and may even vanish; the heart's impulse is greatly diminished in force. The voice becomes husky and feeble. Respiration is shallow and usually labored, varying greatly in frequency. The patient is apathetic, but the mind may be clear to the end.

Recurring cramps of the toes, calves, thighs, hands and arms are generally present, and constitute a very characteristic symptom, for, though present in other diseases, they are more constant and more severe in cholera than in any other, and are a prominent source of suffering.

The urine is very scanty, and may even be suppressed. This suppression is often complete, the bladder remaining empty for days. Death often occurs late on the first day of the disease, and is preceded by profound prostration; or a reactionary stage is developed, during which the vomiting and diarrhœa gradually cease, the collapsic symptoms disappear, and the urine is again secreted, although it is often albuminous and contains blood and tube-casts. The stage of reaction is variable in its duration. With some it passes on rapidly to recovery, while in others it is tedious. Relapses with a recurrence of the symptoms of the algid stage are not uncommon.

There are several groups of symptoms of typhoidal type which may appear at this period of the disease. These are included by most writers under the head of "cholera typhoid." It seems inappropriate to apply this term to so many differing groups of symptoms present in the typhoid state as we meet it in the various infectious diseases, viz., high temperature, prostration, feeble pulse, dry brown tongue, headache, mental dulness, etc. An eruption of an urticarious, erythematous or roseolous type, called by some the choleraic eruption, may appear, especially upon the extremities.

Another type of cholera owes its individuality to nephritis, with



uræmic symptoms, especially of the nervous type. This form is highly fatal. The patient first complains of headache, vertigo, perhaps vomiting. Delirium, passing rapidly into coma, and convulsions may follow.

Besides these forms, various clinical pictures may be drawn, each dependent upon some local inflammatory process. Prominent among them is inflammation of a diphtheritic type affecting the large or small intestine and attended by dysenteric symptoms; and of the pharynx, larynx, bladder, or female generative organs. Pneumonia, pleurisy, bronchitis, peritonitis, etc., have also been observed. Among the most dangerous of the secondary developments are cerebral congestion, merging into meningitis. This latter complication is of the ordinary type, but is often attended by trismus. Less frequent complications or sequelæ are pulmonary œdema, abscesses, suppurative parotitis, gangrene, jaundice, erysipelas, aphthæ, etc.

Cholera runs a very rapid course. The majority of fatal cases terminate in less than twenty-four hours. Cases ending fatally in one hour have been reported, and fatal issues in from six to eight hours are not rare. The stage of reaction, with its secondary developments, may be protracted to two or even three weeks. Mild cases terminate favorably, the patient being soon able to resume work. Fully developed attacks, especially if the reactionary stage is long drawn out by complicating inflammations, may be followed by a most tedious convalescence.

The microscope reveals the comma bacillus in the stools of most cases of cholera Asiatica, but its resemblance to the spirochæte found in cholera nostra is such that cultures are necessary for a differentiation. Gastro-enteritis, poisoning from coal-gas, and the plague, may all present strong resemblances to certain cases of Asiatic cholera.

Certain irritant poisons, notably arsenic, corrosive mercury and ergot develop conditions similar to cholera.

**Prognosis.**—This should be guarded in every case; remembering that the mildest symptoms in the early stage may be simply premonitory of a most violent attack. Probably the symptoms which are most unfavorable are those developing out of embarrassed respiration and failing circulation. The most important of these are cyanosis and low temperature. A sudden attack and a slow convalescence are both unfavorable. Extremes in age conduce to a large mortality, as do also intemperance, debility, overcrowding, poverty and all that that word implies. The mortality of the disease is always great. Of the typical cases fully from one-half to two-thirds die. The mortality in Hamburg in 1892 was 43 per cent. Two-thirds of all the deaths occur during the algid stage.

**Treatment.**—The prophylaxis of cholera is the first question for consideration. The value of strict quarantine has been established. It has prevented the entrance of the disease into the United States since

1873. Next in importance to a barrier to the entrance of the disease, is a limitation of the resulting epidemic should quarantine measures fail. We now have sufficient evidence to demonstrate that an abundant supply of pure water is the most important preventive factor. Cities which cannot meet this demand must expect a high mortality.

Ferran, of Spain, has attempted to secure a protective virus, and the accounts of his experiments claim success; but a French commission appointed to investigate his method did not report favorably upon it.

In 1831 Hahnemann advised the use of copper as a preventive of cholera. Its employment has been quite general, and favorable testimony as to its prophylactic value has come from sources other than homœopathic. Eighteen years after Hahnemann's suggestions, Dr. Burq, of Paris, in the course of investigations as to the therapeutic value of metals, observed that copper-workers possessed a remarkable immunity from cholera. In order to secure the supposed prophylactic virtues of copper, triturations of the pure metal have been used internally. Rings, chains, plates and insoles made of this metal have been worn, and good results claimed therefrom. There can certainly be no objection to this practice, and any virtue it may possess should be taken advantage of.

Cleanliness of person, habitation and soil are of the very greatest importance. During epidemics all water should be boiled and all food thoroughly cooked. *The digestive tract should be kept in the best condition possible.* Every disturbance, however slight, must be treated promptly and thoroughly. The stools and the linen worn by or used upon the bed of the patient should be carefully disinfected. This should be attended to immediately, as the vitality of the germ is less at that time than subsequently. As cholera is but slightly contagious, attendants need have but little fear of contracting the disease directly from the patient if proper precautions are observed.

GENERAL TREATMENT. The patient should be put to bed with the appearance of the earliest symptoms, and kept as quiet as possible. The prompt adoption of efficient measures of treatment is of the greatest importance, resulting in a checking of the attack in some, and subsequent mildness in others. During an epidemic it is, therefore, wise to treat with great care all cases of apparently simple diarrhœa, and any form of disorder of the gastro-intestinal tract. It is considered best to withhold food, or to give it with the utmost caution, until the symptoms are well controlled. If there is abdominal pain the abdomen should be covered with flannels rung out of hot water, and frequently changed. A large mustard plaster applied to the epigastric and abdominal regions is often of service in relieving vomiting and pain. To assuage the intense thirst, cracked ice, iced champagne, or carbonized waters are useful; they also favorably influence the vomiting. Stimulation of the surface of the body and limbs, by means of friction, kneading and brisk rubbing with

mustard water, tends to relieve the cramps and stimulate the circulation. Hot baths of the extremities, or the general hot bath, is called for if the patient is not relieved by these measures. The bath should be commenced at 100° F. and increased gradually to 105° F., and a temperature of even 108 to 110° F. is recommended. The duration of the bath should be from fifteen to twenty minutes, if the relief secured is decided; if but temporary, the bath may be repeated a number of times. Care must be exercised to avoid fatigue on the part of the patient. In favorable cases the cramps are soon diminished, and the general condition of the patient made more comfortable. The influence of the hot bath upon the heart and circulation is also most acceptable, a feeble heart being stimulated and the sluggish blood current accelerated.

During the reactionary stage the patient must be kept thoroughly at rest, liquid food given in small quantities and of the most unirritating character possible. The skin should be kept in good condition, and the patient not allowed to rise to stool.

The remarkable publication by Hahnemann, in 1831, recommending *cuprum* as a prophylactic, has already been referred to. In the same paper its author recommended the remedies which were to be of use in the then approaching epidemic. These have likewise been the most valuable remedies in cholera to this day. They were *camphor*, *cuprum* and *veratrum album*. We have since added only one remedy of equal rank—*arsenicum*.

PREMONITORY PERIOD. For the early diarrhœa and attending symptoms, a variety of medicines may prove useful according to the symptoms present, the most prominent being *ipecac*. Hahnemann advised the immediate administration of *camphor*, however, upon the first appearance of symptoms suggesting cholera, recommending that it be given in drop doses on sugar every few minutes, and at the same time that it be rubbed on the surface of the body. If patients are unconscious or deglutition difficult, then the drug should be evaporated from a hot iron surface, or injected into the bowel with warm water, or both.

*Cuprum* should be selected for the stage of full development, *i. e.*, when diarrhœa, vomiting, cramps, and failing circulation appear. The value of *veratrum album* for the same group of symptoms, to be used alone or in alternation with *cuprum*, was also advanced.

*Bryonia* and *rhus*, Hahnemann thought, might prove to be the remedies for the typhoid symptoms which arise subsequently to the algid stage. The value of the treatment outlined by Hahnemann has been testified to by all physicians who have made careful trial of it, even to this day.

In addition to these Hahnemannian remedies, there is much reason to suppose that the *arsenite of copper*, which has proven so valuable in sporadic cholera, and acute affections of the gastro-intestinal tract, will



be found also a useful remedy in Asiatic cholera. This drug has also been recommended as a preventive. Dr. L. Salzer, of Calcutta, in his monograph on cholera, states that he has used *ricinus* successfully in different epidemics; he also commends it as the most valuable remedy for choleraic diarrhœa. The third dilution was employed. Sircar, also of Calcutta, claims to have obtained excellent results from *hydrocyanic acid* in cases characterized by rapid collapse. Holland, of Bath, England, has observed favorable results from *jatropha* in the third decimal dilution, even after the failure of *veratrum album*. For the reactionary stage we may add to the remedies suggested by Hahnemann, *baptisia*, and the mineral acids.

Should the suppression of the urine be persistent, *stramonium* may be considered, or a class of medicines more closely related to nephritis, viz., *cantharis*, *rhus toxicodendron*, or *terebinthina*.

The rapid loss of serum occurring in most cases of cholera, soon gives rise to a characteristic group of symptoms which, thanks especially to the observations of Cantani, of Naples, we are enabled to combat more successfully. This treatment consists essentially in supplying to the circulation, as rapidly as possible, the water of which it has been deprived. It is accomplished in two ways: first, by the injection, high in the intestines, of water; second, by the hypodermatic injection of water. According to Cantani, the intestinal injections (entero-clysis) are accomplished by means of a long, rectal tube. To secure its retention, the fluid should be slowly injected. If expelled, it serves the purpose of an intestinal irrigation which is advantageous, but the injections should be persisted in, and are usually retained. For an adult, from one to two quarts are recommended. According to Cantani, the injection should consist of an infusion of chamomile, containing five to twenty grammes of tannic acid, twenty to thirty drops of laudanum, and twenty to fifty grammes of gum arabic. The liquid should have a temperature of 100° to 104° F. The injections are to be repeated four times a day, and, in grave cases, after each evacuation. Several observers claim 100 per cent. of recoveries from the adoption of this method in the early stage.

In the later or algid stage, the intra-venous or hypodermatic injection of artificial serum is indicated (hypodermo-clysis). The latter method is most to be recommended. It is accomplished with a fountain syringe, to which is attached a perforated needle. Antiseptic precautions must be observed. The artificial serum consists of chloride of sodium, four grammes; carbonate of sodium, three grammes; and sterilized water, one litre. This fluid is to be injected into the subcutaneous connective tissue at a temperature of 104° F.

The same observer (Cantani) advises that, during the stage of typhoid reaction, the solution for intestinal injection should be composed of a 10 or 15 per cent. solution of chloride of sodium, in sterilized water, to overcome acidity of the blood.



The influence of this treatment is described as most remarkable. Even in well-developed collapse the patient is often aroused, the symptoms gradually diminish, and the patient recovers.

With our lack of experience, it is probably unwise to suggest alterations in this method, but the writer is impressed with the thought that its special value depends upon the introduction of a saline solution into the blood, rather than upon the action of the drugs added to the fluid which is introduced into the intestines. These can act but indirectly upon most of the intestinal surface and contents, and he should feel inclined to first employ simple saline solutions, both for intestinal and hypodermatic use, in association with the general and medicinal treatment indicated for each case.

Nearly all old-school authors advise opium in some form. In the estimation of most, an opiate constitutes the most important treatment of the early stage, and later, if demanded by the control of the vomiting, pain, etc.

Such antiseptic drugs as *naphthalin* and *salol* have been recommended for the intestinal fermentation and diarrhœa, but there is not much evidence of their value in this direction.

## DIPHTHERIA.

**Nomenclature.**—The term diphtheria is now so thoroughly established in medical and even in general literature, that it is unnecessary to mention the numerous obsolete synonyms.

**Definition.**—Diphtheria is a highly contagious disease characterized by a peculiar fibrinous exudate upon mucous surfaces, especially upon those of the pharynx and contiguous parts, and probably dependent upon the infection of a specific bacillus, strong evidence in support of such claim having been advanced.

**History.**—Notwithstanding that accurate descriptions of diphtheria are of comparatively recent date, it is certain that this disease existed in remote antiquity. In the sixth century B.C., D'havantare, in a work written in Sanscrit, described an affection which he called "closing of the throat," and which, judging from his description, was almost certainly diphtheria. Aretæus, in the first century of the Christian era, described diphtheria of both the fauces and the larynx, giving it the names *ulcera Ægyptiaca* and *ulcera Syriaca*. Later, Galen described a disease of the throat with expectoration of false membrane. From his time repeated descriptions answering to that of diphtheria appeared up to the fifth century. From the latter period until about the year 1600 literature does not record anything respecting diphtheria of especial interest. During this long period there is no doubt that the disease prevailed from time to time, and that many of the epidemics of "plague" were really epidemics of the dread disease now under study.

Epidemics were recorded as infesting quite a number of countries towards the close of the sixteenth century. It was very prevalent in Holland in 1557, and was described as "*angina maligna contagiosa*;" and again in 1564 and 1576 and described as "*angina maligna*." An epidemic appeared in Paris in 1576. A great one raged in Spain from 1583 to 1618, the disease there being known as "*garotillo*." An epidemic of malignant sore throat prevailed in Naples from 1610 to 1617. By the end of the seventeenth century the disease established itself in America, and it was during the previous part of this century that it became prevalent in France and Great Britain. The first description of the disease as it appeared in the United States was written by Dr. Wm. Douglas, under the title "*The Practical History of a New Epidemical Eruptive Miliary Fever with Angina Ulcusculosa, which prevailed in Boston, New England, in the years 1735 and 1736.*" A really classical description of diphtheria was presented to the American Philosophical Society by Dr.

Samuel Bard, in 1771. Beginning with 1821 and ending with 1855 Bretonneau presented his studies of diphtheria—studies that have gone far towards improving our knowledge of this disease. It was he who first so fully described the characters of the pseudo-membrane, and showed that whether it appeared in the mouth, the nasal cavities, the throat or the larynx, it produced one and the same disease. This he called “diphtherite.” The work thus begun by Bretonneau has been continued by Trousseau, Louis, Virchow, Mackenzie, Guersant, and Gendron. The bacillus of diphtheria was discovered by Klebs and Loeffler, and is now regarded as a valuable means of differentiating the true from the non-diphtheritic affections.

**Etiology.**—The essential cause of diphtheria is infection with the specific germ of the disease. This may take place either by contagion or inoculation. While then the disease may be carried directly from the sick to the well, the required communication is closer than that in the case of scarlet fever, measles, variola, and certain other contagious diseases. The contagious element is given off mainly in the pharyngeal discharges, and but slightly in the breath of the patient. It appears to possess great vitality and longevity, attaching itself to furniture, walls, clothing, domestic animals, etc. In some cases, the disease seems to have been carried by infected food and drink, as milk, water, etc. Some epidemics of diphtheria appear to have been due entirely to a contaminated milk supply.

Diphtheria may exist either endemically or epidemically. It may be said to be endemic in most communities at the present day. As to the influences which cause it to assume epidemic form, we can hardly be said to know very much. We know that some epidemics are more severe and fatal than others; and that certain of them present symptomatic peculiarities.

Age is undoubtedly a predisposing cause, young children being far more liable to contract the disease from exposure, than those of more mature years. Thus, of 14,688 cases of diphtheria dying in New York between 1873 and 1882, 3,212 were between five and ten years of age, and 10,836 under five. At the same time it must be remembered that no age is exempt, cases occurring even in the aged. The susceptibility of children is explained by the delicacy of their mucous membrane and the readiness with which they respond to irritating influences.

Personal predisposition is exhibited by certain families, who not only suffer once, but several times from exposure.

Climate, temperature, and season seem as a rule to have but little influence as predisposing causes. Cold and dampness, by producing catarrhal affections, put the system in a favorable condition for contracting the disease, and not infrequently are important predisposing causes.

Unsanitary dwellings, bad ventilation, and inefficient plumbing, may be causes in certain cases and epidemics.



The transmission of diphtheria by inoculation is shown by the cases occurring in surgeons who contract the disease from blowing out the contents of a clogged tracheotomy tube, or who become infected from the deposit of discharges on wounds or abrasions.

Diphtheria is auto-inoculable. The patient with pharyngeal diphtheria may infect a wound on himself by permitting the discharges from his throat to come in contact with the same.

One attack affords but a slight and short-lasting immunity from subsequent attacks of the disease.

The Klebs-Löffler bacillus is now regarded as the specific germ of diphtheria. It is found in the outer and middle layers of the false membrane; while from the inner or fibrinous layer it is absent, or is found there in small numbers. In shape it is straight or slightly curved, sometimes spindle-shaped, and sometimes presents clubbed extremities. Its length is about the same as that of the tubercle bacillus, *i. e.*, 2.5 to 3 $\mu$  long; but is about twice as broad as that micro-organism. It is readily destroyed by a temperature of 50° C. It is non-motile, and grows readily on all media between the temperatures of 20° and 40° C. On gelatin plates it forms little round colonies, visible under low powers. Stab cultures show small white drops along the needle track. On potato (alkaline surface) a grayish layer is formed in forty-eight hours. It grows luxuriously in Löffler's blood-serum preparation. This latter consists of blood-serum 3 parts, and bouillon 1 part; the bouillon contains peptone 1 per cent., chloride of sodium  $\frac{1}{2}$  per cent., and dextrin 1 per cent. On this medium it forms a thick yellowish-white layer. It is best stained by Löffler's methylene-blue, which is prepared as follows: 30 cc. of a saturated alcoholic solution of methylene-blue, and 100 cc. of a 1-10,000 solution of potash. The preparation is then washed in a  $\frac{1}{2}$  per cent. solution of acetic acid for from five to ten seconds.

The bacilli *per se* are believed to be incapable of producing any pathological condition. They grow with difficulty, if at all, on healthy mucous membrane. They are always associated with other bacteria, as streptococci and staphylococci. It is believed that the latter—they are often spoken of as the accessory bacteria—first exert their specific action on the mucous membranes, thereby producing a soil on which the Klebs-Löffler bacillus will thrive. The damage exerted by the specific bacillus of diphtheria is through a poisonous product, a ptomaine or tox-albumin. This may be separated from cultures of the bacilli by filtration through a porcelain filter. Inoculated into animals capable of contracting diphtheria, it produces every symptom of that disease. The bacilli themselves are never found in the blood or tissues, other than at points of infection.

A tox-albumin has been separated from cultures of the bacillus, which, if injected into animals, results in nephritis and paralysis

(Brieger, Fränkel). Injections of cultures of the bacillus into the trachea of animals has resulted in a well-developed exudate of the diphtheritic type containing the bacilli, and involvement of the associated lymph-glands, the latter containing extensive necrotic tracts.

The specific product of the bacillus is of a proteid character. It is soluble in water, but precipitated by alcohol. If separated and examined in its purity it is found to be white, amorphous, and an intensely poisonous substance. Animals inoculated with it develop the symptoms and appearances of diphtheria. Its simple application to the healthy mucous membrane produces little or no effect.

The usual cultivation method for the identification of the Klebs-Löffler bacillus is by detaching a small fragment of the exudate, soaking it for a few minutes in a weak solution of boracic acid (2 or 3 per cent.), then applying the membrane to the surface of the blood-serum medium, contained in a test tube, which should be kept at a temperature of 37° C. for about twenty-four hours. If the bacillus is present, small white granules will develop along the track of inoculation. If pure cultures are desired, it is necessary to cultivate two or three generations.

The diphtheria bacillus is readily destroyed by boiling, and survives temperatures considerably less than that of the human body. Its longevity is great, and like the bacillus of tuberculosis it is not destroyed by dessication. Numerous instances have been observed of definite preservation of the poison for years with immediate activity under favorable circumstances. The germ is destroyed by carbolic acid in strength of 1-50, or in mercuric chloride 1-8,000.

While it has been almost certainly determined that the Klebs-Löffler bacillus is the ultimate cause of diphtheria, there are some collateral points still awaiting elucidation. A bacillus to all outward appearances identical with the specific diphtheria bacillus has been found in the mouths of healthy children. Roux and Yersin believe this micro-organism to be the Klebs-Löffler bacillus deprived of its virulence. Inoculations made with it give negative results.

The exact role played by the accessory bacteria is not yet known. It is, however, believed, that their presence in greater or less numbers exercises a very important influence in determining the severity of the disease. We do know that they can affect healthy mucous membranes, and that they are capable of reaching organs and tissues which are not accessible to the specific bacillus of diphtheria. It is certain that they are the cause of many of the associated inflammations and suppurations, notably the aspiration of broncho-pneumonia, and the inflammations of serous membranes.

In view of what has been said of the specific bacillus, and the associated germs, inoculation experiments, etc., it is readily inferred that infection is probably by way of a damaged mucous surface, and rarely

a denuded cutaneous area. If this is true, the prophylactic importance of attention to affections of the mucous membrane lining the throat and related passages is obvious. The germ readily finds its way through the atmosphere surrounding the patient, and is inhaled by attendants, or attaches itself to near objects, from which it is detached later, mixing with the dust of the room; or, is transported to other portions of the house and even to a distance.

As in typhus fever, the "striking distance" of diphtheria is not great, probably not more than a few feet, although much depends upon the ventilation of the apartment. Portions of pseudo-membrane are frequently coughed into the faces of attendants, or into the mouth, nose or eyes, with resulting inoculation. The old idea that the poison reached the blood by means of the lungs, producing first its constitutional influences, is not supported by modern investigation. There seems to be no doubt but that the micro-organism of this disease is inhaled, propagating in the secretions of the throat and finding a lodgment in the tissues of the tonsils or some portion of the mucous membrane lining the pharynx or larynx, or related mucous surfaces.

Recent observations suggest that one of the sources of infection is the convalescent from diphtheria, Roux and Yersin having found the bacillus of diphtheria in the secretions of the throat as late as five weeks after recovery. This suggests greater attention to the disinfection of the affected mucous surfaces after recovery. Beverly Robinson suggests the continuation of disinfectant gargles. The writer does not consider this as at all sufficient, gargles being too limited in their action. Sprays applied to the nose and naso-pharynx are just as essential. For self-protection, as well as the protection of others, physicians should make short visits, and before entering the sick-room cover the entire body and limbs with a blouse, as recommended by Grancher, first removing the coat. An admirable garment for this purpose is a long linen travelling duster. In making examinations of the throat, sitting or standing in front of the patient should be avoided for fear of contamination by means of coughing efforts. The nose and mouth should be covered, as instructed later. Before leaving the house the exposed portions of the body should be washed with an antiseptic solution.

**IMMUNITY.** The experiments of Behring, Fränkel and others have demonstrated the possibility of developing in animals immunity to this disease. The observations of Behring are among the most interesting yet published upon this subject. This observer has been able to produce the disease in varying intensity, developing, mild, severe or complicated cases at will. The same observer has also succeeded in the cure of guinea-pigs suffering from diphtheria by means of the injection of the blood of immune animals.

**THE DISEASE IN ANIMALS.** It has long been supposed that certain of



the lower animals were attacked by diphtheria. The discovery of the Klebs-Löffler bacillus in the exudate has confirmed the popular idea. Diphtheria occurs frequently in cats, fowls, doves, and in some of the larger animals, especially cows, calves, pigs and sheep. It is probable that from cats, in whom the disease attacks the bronchial tubes and lungs, the disease is often communicated to man. Milk infection by means of eruptive changes upon the udder, with resulting infection of the human species, is claimed. A micro-organism supposed to be the exciting cause of diphtheria in fowls and certain other of the lower animals has been described by Roux and Yersin. The conditions favoring the development of diphtheria in animals are filth and all unsanitary conditions.

Within recent years, it has been discovered that there are two varieties of pseudo-membranous inflammation of the throat: one produced by the Klebs-Löffler bacillus, and which may be called diphtheria, and the other, a much milder disease, produced by a bacillus simulating the other, and is thus far known as pseudo-diphtheria. The distinctions between the two will be as fully considered as our present knowledge will permit, in the section on diagnosis.

**Morbid Anatomy.**—The most important lesion of diphtheria is an inflammation of the pharynx and often of the communicating cavities, attended by the development of a membranous exudate. The usual order of development of the lesion is as follows: A grayish or whitish spot or spots of membrane appear upon one or both tonsils, resting upon an inflamed base; or a more or less general inflammation of the throat may precede the appearance of the diphtheritic exudate. The areas of membrane enlarge by peripheral spreading and finally coalesce, covering more or less of the tonsils. Similar development of exudate may appear primarily upon the soft palate or back of the pharynx, although these regions are usually involved by extension. With the progress of the disease—within a few days, if the attack is an aggravated one—extension to the larynx, nasal chambers, or mouth, may take place. Involvement of the œsophagus has rarely been noted; probably it would be observed more frequently did this complication lead to more serious symptoms than it does. Stenosis of the larynx with all the phenomena that this entails is the result of extension to that organ. A number of post-mortem examinations that I have made, especially upon tracheotomized cases, has shown that the obstruction is due more especially to thickening of the intra-laryngeal tissue rather than to an accumulation of exudate in the narrow portion of the organ.

Extension to the nose occurs in quite a number of cases and leads to swelling, obstruction of the nasal passages, with characteristic discharges. The membrane is usually grayish and fills the nasal chambers; it may even extend externally to the lip.



Much of the buccal cavity may be involved. It is not rare for the interior surface of the soft and hard palate to be covered with membrane. The uvula is often much enlarged by reason of œdema.

The lymphatic glands at the angles of the jaw are swollen; sometimes enormously so.

Any of the mucous membranes, viz., the conjunctiva, that lining the bronchi, œsophagus, stomach, biliary ducts, the intestines, or vagina, may have the false membrane developed upon it. Even the skin if wounded or denuded of its epithelium may become covered with exudate.

To Oertel, more than to any other pathologist, is due the credit of making clear the microscopic peculiarities of this affection. The inflammatory change in diphtheria manifests certain variations in development which correspond to clinical varieties of the disease. Certain mild cases may be attended by more or less denudation of the surface epithelium, or there may be all the pathological evidences of a catarrhal inflammation; or, in the characteristically developed lesion there is a fibrinous exudate infiltrating the mucous membrane and submucous tissues. This exudate, in combination with epithelial cells, pus cells, blood cells, debris, and bacteria of various kinds, forms a membrane varying in thickness from a mere film to a quarter of an inch.

The coagulation necrosis of Weigert occurs in certain areas, both in the false membranes and within the mucous tissues. This condition, it will be remembered, consists of a hyaline metamorphosis of cells. It is believed, by Oertel especially, that the primary influence of the diphtheritic poison is to produce such a necrosis of, first, the superficial cells of the mucous membrane, and later, of the deeper cells of the mucosa.

With age the membrane grows darker and foul, and emits a stench. It is removed by disintegration or exfoliation, pus forming beneath the membranous layer. In the process of exfoliation the edges curl up and in from two to five or six days it is thrown off *en masse*. High degrees of phlegmonous inflammation of the entire or localized portions of the throat may set in, and abscess or œdema may occur as complications.

Bronchitis and lobular pneumonia are often developed at the height of the disease, and the glandular organs generally present a parenchymatous degeneration. Hæmorrhagic points are common in the serous membranes; indeed they have been noted in very many of the organs and tissues.

The cavities of the heart are often dilated and may contain thrombi. The cardiac muscle is often fatty.

The paralysis of diphtheria is due in most instances to changes in the peripheral nerves. There are some clinical and pathological data which show that the spinal cord is occasionally involved. Our knowledge of this subject is due largely to the investigations of Meyer and Leyden.

In malignant cases the blood is altered as much as it is in the acute infectious fevers.

**Clinical Course.**—The time intervening between exposure to the contagion and the development of the first symptoms is by no means definite. It has been variously stated by authors, the extremes being one day and three weeks. From one to two weeks is usually regarded as the ordinary duration of the period of incubation. In cases in which the disease has been transmitted by inoculation this time is much shorter, *i. e.*, from one to four days. The stage of incubation varies in duration in different epidemics. In but very few cases can it be accurately determined. The time of exposure does not necessarily correspond with that of infection, for the bacillus may be taken into the throat, lodge there, remaining for several days before the tissues are in a suitable condition for it to propagate.

The symptoms of diphtheria may be epitomized as follows: (1) A febrile attack; (2) sore throat; (3) systemic infection. To these may be added the symptoms resulting from complications or extensions of the disease.

Slight prodromata may exist for a day or two before the advent of any local manifestation. This fact is regarded as possessing some diagnostic value in differentiating diphtheria from certain benign throat affections, which are usually of more rapid onset. The symptoms of invasion are mainly such as accompany many ordinary febrile attacks and are general pain, headache, anorexia, nausea, vomiting, vertigo and constipation, and finally the symptom which first directs attention to the true nature of the malady—sore throat. These initial symptoms are sometimes very mild, sufficiently so to either escape attention altogether, or to permit of the child remaining out of bed. The temperature in diphtheria is seldom high, ranging usually from 100° F. to 103° F. It may exceptionally rise to 105° F. Pyrexia must not be regarded as an important symptom; its severity is of but little use as a prognostic indication. In very young children the disease may be ushered in by a convulsion.

The condition of the throat is the most important feature. Acute soreness is usually complained of. The physician must remember, however, that this is not necessarily the case, for oftentimes the pain is so slight as to give the little one no inconvenience. Inspection of the parts is therefore necessary. First there is seen a hyperæmia of the tonsils, soon extending to the pillars of the fauces. This congestion may be of a more mottled character than is observed in the ordinary catarrhal inflammations about the throat. Shortly there follows an exudate upon the tonsils. It begins as yellowish or grayish spots, which coalesce and spread to the fauces, soft palate, pharynx, etc. By the third or fourth day, the membrane covers all these parts. The odor of the breath now becomes more and more offensive; swallowing grows painful; the lymphatics at the angle of the jaw swell, and the patient manifests every

evidence of a serious illness. The pulse becomes rapid and weak, ranging in frequency from 120 to 140 per minute. There are great prostration and perhaps some slight albuminuria. These latter symptoms are both regarded as of diagnostic importance accompanying throat affections of doubtful character. By many the albuminuria is thought to be of very frequent occurrence. The symptoms above enumerated continue until about the close of the first week, when, if the course of the disease is to be favorable, there is an improvement in the general condition. The membrane now gradually exfoliates and disintegrates, swelling of the lymphatic glands diminishes, and by the middle of the second week the throat is clear and the patient well entered upon convalescence seldom marred excepting by paralysis. If the course of the disease is to be unfavorable, the strength diminishes, the lymphatic glands become greatly enlarged, the breath putrid, the membranes darkened by the accumulation of foreign matters, and decomposition changes. Swallowing may grow very difficult, or regurgitation of fluids and even of solids through the nose, may occur. The causes leading to unfavorable results are: (1) systemic infection; (2) extension of the disease to the nasal cavities or larynx, and (3) involvement of the bronchi and pulmonary tissues. The disease may appear primarily in either the nose, larynx or bronchi,—very exceptionally indeed in the last named. Bronchial involvement is usually secondary to diphtheria of the larynx and trachea.

**NASAL DIPHTHERIA.** The symptoms attending the development of nasal diphtheria can be easily surmised. They are substantially the same, whether the nasal affection is primary or secondary to the pharyngeal form. Obstruction of the nasal passages, an acrid and offensive yellowish discharge irritating the upper lip, prostration, breathing with the mouth wide open and marked glandular swelling are practically always present. Epistaxis is frequent. The glands involved are the submaxillary and those at the angle of the jaw, these having intimate connection with the rich lymphatic supply of the nose. In some few cases, the glandular swelling is the initial symptom to appeal to the observer. The disease sometimes extends through the lachrymal ducts to the conjunctiva; and in others through the Eustachian tubes to the middle ear, producing otitis media and deafness.

**LARYNGEAL DIPHTHERIA.** This is usually an extension of the disease from the pharynx, although it too may be a primary manifestation of the action of the diphtheritic poison. Under the latter circumstance, it resembles membranous croup very closely. The weight of medical opinion just now seems to favor the identity of these two processes. Laryngeal involvement may occur in connection with any degree of pharyngeal diphtheria, although it is most frequently associated with extensive membranous exudation. The symptoms are croupous cough and gradually increasing cyanosis from stenosis of the larynx. The



cough is sometimes paroxysmal from functional spasm of the vocal cords. The cyanotic symptoms vary as the obstruction is of rapid or slow formation. When the former, blueness of the surface is observed; when the latter, pallor or earthy hue predominates. Evidences of the obstruction are seen in the soft parts of the chest, as the intercostal spaces, which sink in during inspiration. Sometimes the obstruction is mainly expiratory, when the same parts are bulged during expiration. Temporary relief of symptoms is occasioned by expectoration of false membrane. A new exudate is rapidly formed, however, sometimes within the incredibly short time of half an hour. Throughout the cyanotic stage, the patient is restless. Prostration and glandular swelling are but slight in the pure laryngeal cases, as the lymphatic communication between the larynx and the glands at the angle of the jaw is not at all free.

EXTENSION TO THE BRONCHI AND LUNGS cannot always be clearly diagnosticated. Physical examination of the chest gives some help, especially in relation to the lungs. Areas of pulmonary consolidation are not uncommonly discovered towards the close of the attack and add greatly to the gravity of the case. A sudden rise in temperature, rapid respiration and marked dyspnoea, must be regarded as very suggestive.

SYSTEMIC POISONING. Evidences of profound infection exist in most cases of diphtheria which pass on to a full development or to a fatal issue, and that irrespective of the distribution of the lesions in the pharynx, larynx or nasal cavities. The mildness of the local manifestations is sometimes a very poor guide as to the character of the general symptoms, for in many so-called mild cases there appear early great prostration, an exceedingly rapid and feeble pulse, serious nervous symptoms, sometimes petechia and bleeding from the mucous membranes, and perhaps a high or a subnormal temperature. The patient sinks overpowered by the action of the poison on the blood and nerve centres and dies comatose or from failing heart.

The skin eruptions of diphtheria are generally ascribed to the general infection by the accessory bacteria. They are not essential phenomena of the disease. They may be of different types. The most common is a general *erythema* resembling that of scarlatina, but differing from the eruption of that disease in the absence of desquamation. Another and more severe eruption is *purpura hæmorrhagica*.

Several varieties of diphtheria have been mentioned by writers, the classification depending upon the intensity of the disease and locality of the exudate. The terms designating them are self-explanatory and need only to be mentioned to be understood. They are: (1) Catarrhal or mild diphtheria; (2) typical diphtheria; (3) malignant diphtheria; (4) chronic diphtheria; (5) nasal, pharyngeal, laryngeal, etc., diphtheria.

**Complications.**—Diphtheria may itself be a complication of any disease, notably, however, of such as scarlatina or measles, in which the

morbid changes predispose the system to the lodgment and propagation of the bacilli.

On the other hand there are a few conditions which from their relative infrequency may be spoken of rather as complications or sequelæ than as actual clinical incidents of the disease. These are during the attack, pneumonia, nephritis, hæmorrhages. *Pneumonic processes* frequently occur, but escape attention until the patient dies and an autopsy reveals their presence. Capillary bronchitis is shown sometimes in fatal cases. The failure to recognize these thoracic complications during life is due undoubtedly to our neglect of physical examinations and the overshadowing prominence of the symptoms relating to the throat and general system.

*Nephritis* is rare, although albuminuria is present in all aggravated cases. The amount of albumin may be large, though if the result is favorable, the albuminuria disappears gradually with the general improvement. Microscopic examination reveals the presence of epithelial and granular casts. The nephritis of diphtheria differs clinically from that of scarlatina in the infrequency of dropsy as a symptom. Its seriousness depends upon the stage of the disease at which it is developed. The later its development, the more difficult it is of control. It usually runs a course of a week or two. Occasionally it becomes chronic.

Hæmorrhage from the nose or throat is not uncommon, and may occur rarely from other points. In most instances it is attendant upon ulcerative changes. In malignant cases it is an annoying symptom, and in a case recently seen, probably precipitated the fatal issue.

*Paralysis* is a dangerous and not infrequent feature of fully developed diphtheria, as well as its most important sequela. It has now been pretty well determined to be the result of ptomaine intoxication. Of the paralytic developments, that of the heart is the most important. It may occur at any time after the full development of the disease, or be delayed until convalescence seems to be thoroughly established, and may continue for weeks after the disappearance of other symptoms. This cardiac weakness during the course of the disease bears no regular relation to the intensity of the primary affection. It may be severe in the mildest type of diphtheria. I can recall a case in which the pulse was thready and beating 160 to 180 per minute on the fifth day of the illness, the child apparently being so slightly sick as to sit up in bed and amuse itself with its toys. Sudden death is very common in such cases, usually following some undue exertion. Even with an improvement of the cardiac weakness, and the disappearance of all the symptoms of diphtheria, danger still lurks. Only recently I saw with Dr. Chas. E. Myers, of this city, a boy aged nine years, who after one week's illness developed a high degree of heart-weakness controlled only by the most painstaking treatment. The patient then passed on to uninterrupted

recovery, but died within six hours of the onset of a second attack of heart-failure. During the interim between the two attacks, the pulse was not apparently weaker than during convalescence from any debilitating disease.

An abnormally slow pulse—bradycardia—must also be looked upon with anxiety. Some assert that such cases augur unusual danger.

A more frequent form of paralysis, but one less serious, is that which affects the throat, usually attacking the soft palate. It does not often develop until the original throat symptoms have been well for a week or two. Upon examination the *velum palati* will be found relaxed and its sensation variously impaired. If one side only is involved, the curtain is drawn to the opposite direction; the pharyngeal mucous membrane is usually more or less anæsthetic, and the function of deglutition impaired. If there is a post-diphtheritic pharyngeal catarrh in these cases of paralysis, the pharynx is sometimes seen covered with a frothy discharge resembling the ebullition produced by the application of hydrogen peroxide. The voice acquires a nasal tone and liquids regurgitate through the nose, these being the symptoms which first suggest the presence of the paralytic throat trouble. Within a few weeks the complication generally disappears. It may, however, form a part of a general affection of the nervous system, in which case it is decidedly more persistent. When pharyngeal palsy develops, further involvement of the system is suggested by a sense of weariness in the extremities and an impairment of or loss of the patellar tendon reflex, as pointed out by Buzzard. The latter authority even suggests the frequent testing of this phenomenon during the convalescent period, as its absence is a pretty sure forerunner of the paralysis that may come. In some cases, it may be the only tangible evidence of that complication.

Quite a variety of local paralyses may occur, though but rarely: viz., paralysis of any of the eye muscles, leading to loss of power of accommodation, falling of the upper lid, or strabismus of some variety. General paralysis of the voluntary muscular system may occur and is apt to develop subsequently to the pharyngeal paralysis. The lower extremities are oftenest involved, the loss of power usually beginning in the feet. There is, however, no regular order of development; all the limbs may be entirely helpless, the patient being obliged to recline, or the loss of power may be only sufficient to lead to pauses and uncertainty in movements. If there is paralysis of the sphincters, involuntary discharge of *fæces* and urine will occur. The genitals are sometimes involved, resulting in loss of sexual power for a long period. The muscles of the trunk may be involved later, as shown by the interference with respiratory movements. Unsteadiness of the head indicates a palsied condition of the muscles of the neck.

The laryngeal muscles are seldom affected excepting in association



with the general paralysis. The voice then is altered and may be lost. There is marked dyspnœa. If the superior laryngeal branch of the pneumogastric is involved, loss of sensation ensues, and choking attacks result from the passage of particles of food into the larynx.

Diphtheritic paralysis may result fatally, though recovery usually ensues. Its duration is very variable. The statement that it is self-limited in duration is a mistake. Slight cases recover within a few weeks; others continue sometimes for months or from one to three years. The paralysis of diphtheria is due to a toxic neuritis. That it is caused by the action of a tox-albumin developed by the bacilli, has been shown by inoculation experiments upon animals.

Inflammation of the serous membranes is a very rare complication.

**Diagnosis.**—The clinical differentiation of diphtheria, and the conditions which simulate it, is sometimes an almost impossible task. There are cases in which the symptoms are such as to make a distinction apart from a bacteriological examination of the pharyngeal exudate an utter impossibility. We are perhaps called upon to separate diphtheria from *follicular tonsillitis*, or *follicular angina* as it is called by some practitioners, more frequently than from any other disease. The problem is especially difficult in the early stages of the affections, just when a correct conclusion is so important. As both diseases are contagious, it is better to isolate the patient, be our opinion what it may, and treat the case as one of diphtheria until a positive diagnosis is made to the contrary. It should not be forgotten that follicular tonsillitis may by infection with the Klebs-Löffler bacillus be succeeded so promptly by a true diphtheria as to give the impression that but the one disease has existed from the inception of the illness. Careful watching of the development of the lesions soon results in the necessary data for differentiation. In follicular tonsillitis the exudate is found to be related to the tonsillar crypts. It is very readily removed by gentle syringing or by careful wiping with a cotton-covered probe. The mucous membrane beneath is found to be uninjured. In diphtheria, the membrane cannot be so readily removed, and when a portion of it is taken away, the mucous membrane beneath is found abraded and bleeding. In this disease also the exudate is beneath the epithelium, through which it subsequently bursts. In follicular tonsillitis, it extends by involving successive crypts. In diphtheria the membrane enlarges by peripheral extension. Extension of the disease process to the surrounding tissues of the soft palate and uvula is indicative of diphtheria. The history of exposure to the contagium of diphtheria is of course a valuable assistance in determining a conclusion. While we should carefully guard against mistaking diphtheria for a simple throat affection, we should be equally careful to avoid the common error of considering every throat affection with exudation diphtheria. Doubtless many of the excellent results claimed for certain

methods of treatment of diphtheria find their origin in this last mentioned diagnostic error. The mode of onset of the illness is sometimes of positive value, as a means of diagnosis. Diphtheria is often preceded by evidences of an oncoming severe illness, the really dangerous symptoms not making themselves manifest for two or three days. Follicular tonsillitis often has a more rapid onset. The child may have been perfectly well during the day; at night it develops fever, and in the morning it has a severe type of "sore throat." This point must be regarded as possessing relative value only.

Of great importance likewise is the differentiation of diphtheria and *pseudo-diphtheria*. The investigations of Park, undertaken to determine whether or not there exist any phenomena apart from those discovered by bacteriological examination, by which these clinically related affections may be distinguished, have failed in great measure of their object, though giving us, as they do, valuable information in other directions. The exudate in *pseudo-diphtheria* is thinner than in diphtheria, and when extending to the other parts of the throat, first attacks those portions of the uvula lying in contact with the diseased tonsils. A bacteriological examination is necessary, however, in order to be positive. Unfortunately this procedure is one involving a degree of technical skill not attained by many general practitioners.

*Scarlatina* sometimes presents a clinical picture resembling that of diphtheria. Careful inspection will, however, show the eruption appearing at the proper stage of the disease, and the inflammatory lesion in the throat is generally of a more phlegmonous character and has less exudate than diphtheria. Later it is apt to be attended by ulceration of the tonsils. When diphtheria is attended by a rash, it is sometimes impossible to make a diagnosis for several days.

*Post-diphtheritic palsy* sometimes closely simulates locomotor ataxia. Diagnostic errors will occur when the existence of a previous throat affection has been overlooked. The mode of onset affords an all-sufficient aid for differentiation.

**Prognosis.**—A guarded prognosis should be given in every case, as many beginning with all evidences of a benign course, later prove fatal. Death may result from systemic infection, laryngeal, lung, or nasal involvement, heart-failure or uræmia. When the membrane is confined to the tonsils, the outlook is generally favorable, for the lymphatic supply of these structures is so limited as to afford less opportunity for absorption of the toxic products of the bacilli. The nasal form of diphtheria is always dangerous. The parts here are rich in lymphatics, and the opportunities for systemic infection are correspondingly great. The conformation of the nasal cavities, moreover, does not favor thorough drainage, and this forms another element of danger. Epidemics differ greatly in their fatality, some destroying nearly all young subjects at-

tacked, while others present but a small mortality. It is not an uncommon experience for practitioners to tell of losing but few cases for years, and the epidemic type changing, they encounter a frightful mortality. The presence of extensive exudation suggests a grave case. Laryngeal and nasal involvement and heart-failure warrant a cautious prognosis. Quantities of albumin in the urine or a parenchymatous nephritis are both of ill omen.

**Treatment.**—THE PROPHYLACTIC treatment of diphtheria is of the highest importance. Greater care respecting the isolation of patients, the destruction of discharges, the keeping of children of the same household from school, and the thorough disinfection of houses in which the disease has occurred, will do much to lessen the spread of the disease. After the recovery of the patient, the entire interior of the sick-room should be scrubbed and then washed with a 1-2,000 solution of bichloride of mercury or other effective germicidal solution. The old method of fumigation with sulphur is of little value. Very important also is the disinfection of the drain-pipes of the wash-stands, bathtubs, and water-closets of the house, whether they have been used in connection with the sick-room or not. The interior of these leaden tubes, covered as they are with grease, form an excellent bed for cultivation of the specific bacilli, and in several instances have seemed to the writer to be for a long time the principal causes of the disease in the house. One family who lost three children in as many years and in whose midst seven cases developed in that period of time, had the disease blotted out, although susceptible material remained, by careful attention to these pipes. Everything else seemed to have been done previously.

Attention has of late been directed to the condition of the mouth and throat of children as a predisposing factor. The most importance attaches to the tonsils. If these organs are large and present numerous crypts, they should be removed, or better, treated with the galvano-cautery. Under the influence of cocaine, a number of punctures of each tonsil can be made without a special degree of pain, at the time or subsequently. Three or four sittings will result in a marked degree of contraction and a prevention of the recurrent attacks of follicular and other forms of tonsillar inflammations, and thus destroy the soil upon which the bacilli of diphtheria love to propagate. Careful cleansing of the teeth, and the use of antiseptic mouth and throat gargles are advocated by some.

**CARE OF THE PATIENT.** The patient should be placed in a light room with the window properly curtained. Only light sufficient to make a cheerful apartment should be permitted during convalescence. Every unnecessary article of furniture should be removed, especially upholstered pieces. The floor should be bare, or at least covered with a rug which can be subsequently destroyed. Outside and inside the door sheets moistened with disinfectants should be hung. Good ventilation, an



absence of drafts, and an even temperature of from 70° to 73° F. are necessary. If the patient's age permits he should be kept at perfect rest in bed. Inability to secure proper quiet is a disadvantage we suffer from in treating young children. The patient must be kept scrupulously clean, especially about the exposed portions, all cloths or cotton used to remove discharges should be burned at once, or put into a vessel filled with a powerful disinfectant and kept handy for that purpose. All dishes used should be passed out of the room into a pan of disinfecting fluid and allowed to remain for an hour or so before being washed in clear water. All bed-clothing used should be treated in the same manner, and no clothing of a heavier character allowed to leave the room until after recovery and fumigation. While working with the patient, the physician had better cover his mouth and nose with a shield of cloth saturated with a disinfectant. Such a shield can be conveniently made by tying a handkerchief about the head just below the eyes. The lower ends can be tied about the neck. It is remarkable in view of the general carelessness in manipulations that more physicians are not attacked by diphtheria.

**Food.** As diphtheria is a highly debilitating disease, the patient should be well fed with the most nutritious food, preferably liquids. A little solid food may be taken in mild cases, and during convalescence. Milk is acceptable to most young patients and answers for food and drink, an important desideratum when swallowing is very painful. The best form for administering milk is koumiss. I consider it very fortunate when patients will take this article freely. As it cannot always be procured, the following formula for its preparation may be acceptable to many readers. Extensive experience with this preparation enables me to highly commend it. To make six pints, heat that quantity of pure milk to 120° F. Add to this one compressed yeast-cake and six teaspoonfuls of sugar well rubbed up with a little of the milk. Bottle at once, not filling the necks of the bottles, in strong patent-stoppered bottles, or in bottles with their corks tied in. Keep at a temperature of 80° to 90° F. for about six hours; then at an ordinary room temperature, 70° F., for about an hour, to secure gradual cooling, and finally place upon ice. The koumiss is ready for use as soon as cold. If it is desired to avoid cold food, the bottle can remain in the room long enough before using to secure the desired temperature.

Farinaceous food long cooked, beef-extracts, the juice from a rare roast or steak, beef-peptonoids, sarco-peptones, etc., may be useful. The free use of fruits answers well in many cases. The juice of oranges, or breadfruit, a little scraped apple or pear, or better than either, well-selected grapes (rejecting the skins and seeds) are very grateful and will often be taken and well borne when other food is rejected.

**STIMULANTS.** The employment of alcoholic stimulation is almost universal in the treatment of diphtheria. It may be brought into requi-

sition as soon as prostration from systemic poisoning or heart-failure is present. The amount given will depend upon the age of the patient and his condition. It must be judged entirely in regard to individual cases. The tolerance of diphtheritic patients to alcoholic beverages will lead to the use of relatively large doses. In a case of naso-pharyngeal diphtheria occurring in a boy of seven years, which I recently treated with Dr. Titman, of Mt. Airy, there was scanty, highly albuminous urine, and failing heart. The favorable result seemed largely due to champagne, which the child took willingly in quantities of one or two pints in the twenty-four hours.

**EXTERNAL LOCAL TREATMENT.** Externally a great deal of comfort can be secured from hot poultices or fomentations, if pain is annoying or especially if the glandular swelling is great. Ice is contraindicated, as it tends to prevent the development of suppuration beneath the pseudomembrane, which in diphtheria is a conservative process.

**LOCAL TREATMENT OF THE THROAT.** All active treatment having for its object the removal or immediate destruction of the exudate is to be condemned. Removal of loosened foul membrane is of course desirable, but it seems certain that all undue irritation of the pharynx from instruments and applications tends to an extension of the diphtheritic lesion. Local internal treatment, however, if judiciously applied, yields most gratifying results. It should be begun at once, viz., upon the first appearance of the throat lesion. If effective, the foul odor is destroyed, the extension of the exudate checked, and the production and absorption of poisonous substances with the consequent systemic infection greatly reduced. As most patients are young children, it is important to adopt a treatment which will be tolerated. The following method, published by me in the *Hahnemannian Monthly*, January, 1891, has been very favorably reported on by many observers. It consists essentially in the thorough application of the permanganate of potash to the affected regions. Sprays were of little use, but a trituration of the drug in the strength of one grain to the ounce, in a mixture of fine sugar of milk and gum acacia, blown over the affected parts every few hours, was found to attach itself to the membranous surfaces and promptly remove odor and lessen discharges. The treatment is of little value unless thoroughly carried out. If the nose is involved, a passage-way must be tunnelled out with cotton and forceps, using cocaine if necessary. Then the permanganate trituration should be blown into every accessible portion of the nasal chambers. This application may be preceded by a spray compounded of equal parts of peroxide of hydrogen solution and water, although I do not always use it as a part of this plan. The resulting froth must, of course, be thoroughly wiped off with cotton. With courage and perseverance on the part of the physician many a life may be saved mainly through judicious early local treatment. Sprays of lime water and lactic acid, papoid, trypsin, and other agents for the solution of the membrane have not resulted very favorably.

Certain of the aniline dyes have attained a reputation in the local treatment of diphtheria. The writer has employed pyoktanin-blue considerably in the early stages, and with very encouraging results. A saturated watery solution was employed. It seems important that the application should be very thoroughly made, and to accomplish this ether has been several times employed. Not only should the stain be applied to the areas covered with exudation, but by means of probe, cotton, and spray, to every portion of the upper respiratory tract. One such application has been followed by an arrest of typical cases in several instances.

Applications to the throat, especially in young children, may be often quite satisfactorily made by dropping the liquid into the nose. If it is desirable to avoid contact with the nasal chambers, the application may be injected through a very small calibered rubber tubing, which can be oiled and passed into the naso-pharynx.

MEDICATION. In the early stages of diphtheria of the mild type, *belladonna*, *mercurius bin.*, and *phytolacca* are in common use and of undoubted value. With the development of severe throat or general symptoms, however, another class of medicines must be considered. The most prominent of these is undoubtedly *mercurius cyanatus*, originally recommended by Dr. Beck, of France, and championed by Dr. Villers, of St. Petersburg. This medicine has been prescribed in varying doses, but the second and third decimal triturations seem to be most in use.

*Mercurius corrosivus* seems, according to the experience of some observers, to exercise a more decided influence than the cyanuret. This statement has been corroborated by the experience of many colleagues, as well as by several authors of the old school. Dr. W. H. Bigler highly commends it. It is best administered in the third decimal dilution, five drops every one to three hours. Some administer doses as large as from the one-sixtieth to the one-twenty-fourth of a grain every few hours. Patients suffering from acute infectious diseases, it must be remembered, often, perhaps usually, tolerate really enormous doses of poisonous drugs, apparently suggesting the necessity for larger doses than many administer.

*Rhus tox.* is indicated especially by extensive swelling of the lymphatics and cellular tissue. Prescribed upon this symptom, it has repeatedly controlled all serious symptoms and transformed a severe case into a mild one. The general well-known rhus symptoms are often present in various stages of the disease, under which circumstances the remedy may be prescribed with confidence.

*Cantharis*. Several observers have recommended this drug when the throat looks as if blistering fluid had been applied and there is much pain on swallowing. The *potash* salts hold a high position in the treatment of diphtheria. The *bichromate* is quite generally employed, espe-



cially in laryngeal cases. I think its reputation in this variety should be greater than it is. For the yellowish exudate it seems especially applicable. I agree with Hughes that "thickness and tenacity of the false membrane will always be an indication for it." Ropy expectoration, a heavily coated yellowish or a cracked and irritable red tongue and an irritable stomach are all valuable indications. Hughes finds this remedy specific in nasal diphtheria.

The *permanganate of potash* has exercised a more beneficial action generally in diphtheria than any other medicine, but this is evidently due to its local action. The exceedingly rapid decomposition of this drug is detrimental to its action as an internal remedy. This very quality, however, renders it more valuable as a local agent, and as already indicated, it is unequalled as a deodorizer and a disintegrator of the membrane. If abdominal pain, offensive diarrhœa, or irritable stomach develop, this medicine is doubly indicated.

The *chlorate of potassium* is but very little used, but it has been found useful in cases of diphtheria showing very rapidly developing anæmia (blood changes) and especially for the complicating nephritis. The very pronounced action of this medicine upon the kidneys warrants its more general employment.

The *carbonate of potash* has seemed to act favorably upon some cases of diphtheria with large, hard swelling of the glands, cases not helped by rhus, biniodide of mercury, etc.

*Hydrochloric acid* has considerable reputation in the treatment of diphtheria among all classes of practitioners. Hughes recommends it in the "lesser degrees of toxæmia." In general it may be said that the symptomatic indications for the use of this drug are the same as those calling for it in typhoid fever.

*Arum triphyllum* has given undoubted evidences of its value in diphtheria; excoriation and glandular involvement seem to be the especial indications. In typhoid conditions, with red cracked tongue and lips, excoriated upper lip and nostrils, nasal involvement and much involvement of the glands and deeper tissues, there is common consent as to its value.

*Apis mellifica* is considered by many of the ablest practitioners as one of the most effective anti-diphtheritic remedies. A study of its action indicates that its homœopathicity is more than local. However, the leading indication is unquestionably the œdema which attends the pharyngeal lesion.

*Lachesis* has been very popular as an anti-diphtheritic in this country. Carroll Dunham valued it highly in diphtheria and found it useful "when the constitutional symptoms predominated over the local," when in association with throat symptoms of slight intensity there was great prostration, a slow, feeble pulse, a cold, clammy sweat, and pain out

of proportion to the local lesion, etc. Gangrenous changes in the throat should always suggest it. Hyperæsthesia of the neck and other subjective symptoms of this medicine must be remembered.

TREATMENT OF THE LARYNGEAL EXTENSION. In laryngeal diphtheria it is common practice to keep the room warm and charge the atmosphere heavily with steam. This may be done by building a canopy or tent with sheets over the bed, placing tea-kettles over Bunsen burners or gas-stoves near the bed, and conduct the steam beneath the canopy by heavy rubber pipes. An equable moist atmosphere is an advantage. If too warm, however, it favors debility and undue sweating. Too often superheated rooms are illy ventilated. The necessity for oxygen in these cases must not be overlooked. There is little indeed to be said on this subject in addition to what has already been said in speaking of the treatment of pseudo-membranous laryngitis. Tracheotomy is often necessary, but the results from this operation are in most cases temporary only. Notwithstanding this, the procedure is perfectly justifiable, owing to the remarkable relief of dyspnœa obtained thereby.

As to remedies, *kali bichromicum*, *bromine* and especially the *chloride of lime* have given the best results in my experience. *Iodine* in drop doses of the officinal tincture, well diluted in water, has seemed valuable when the laryngeal diphtheria was primary. Perhaps it is better adapted to brunettes who have been thin and who have large appetites.

TREATMENT OF POST-DIPHTHERITIC PARALYSIS. Even if the post-diphtheritic paralysis does "always" or nearly always result in recovery, its progress can be accelerated by the use of a well-selected medicine. Of these *gelsemium* has proven the most useful. *Cocculus* is indicated in a widely distributed post-diphtheritic paralysis in which both motor and sensory functions are destroyed. The author has found the *chloride of gold and sodium* 2x apparently useful in the peripheral neuritis of general distribution. Electricity, especially galvanism, is an invaluable adjuvant. When anæsthesia is a marked feature, the application of the faradic brush to the affected areas will be productive of excellent results.

## ERYSIPELAS.

**Nomenclature.**—The rose (Scotland); St. Anthony's fire (England).

**Definition.**—An acute contagious disease caused by a specific streptococcus, and characterized by a spreading inflammation of the skin.

**Etiology.**—It seems to have been satisfactorily determined that erysipelas is due to the introduction of a specific germ, the *streptococcus erysipelidis* (a chain-forming micrococcus), which has been carefully studied by Lakonsky, Oth, Fehleisen, Ziegler, Koch, and others. Its habitat is especially in the peripheral inflammatory zone, *i.e.*, where the disease is progressing actively and minutely, in the lymph vessels and serous canaliculi of the diseased skin and subcutaneous tissue. They may even penetrate these small canals beyond the area of inflamed tissues. Several experimenters have succeeded in cultivating this streptococcus and developing erysipelas by inoculation. Fehleisen has established the question beyond doubt by inoculating patients for the purpose of modifying existing maladies by the development of erysipelas. In the human subject the skin inflammation was produced as early as fifteen and as late as sixty hours after the inoculation. A second experiment at the expiration of a few months resulted in a repetition of the disease. Erysipelas is both traumatic and idiopathic, *i.e.*, it develops in connection with wounds, or upon cutaneous and mucous surfaces which do not manifest evidence of injury of any kind. The traumatic variety occurs not only in connection with well-defined wounds, but in the course of various diseases of the skin, the lesions of which serve as the equivalent to wounds. It is common for it to develop in, and spread from, the umbilicus of the new-born child, and it is not rare as a puerperal condition. Of its highly contagious character in surgical and parturient wards, surgeons and obstetricians are well aware. No better proof of its contagious nature is needed than the marked decrease in the frequency of the disease since the dawn of the antiseptic era in surgery. While it seems clear that erysipelas spreads by inoculation from one open wound to another, it is not certain that the idiopathic cases so originate. The fact that the disease develops most frequently upon exposed surfaces of the body, upon portions which are subject to small and often unnoticed injuries and to contact with flies and other insects which may directly convey the poison, is strongly suggestive that these cases may develop in a poison deposited upon the surface. How far then the so-called idiopathic cases are such, must remain an open question. It is positively asserted that the disease always originates by infection from without;



that it obtains entrance to the system by some abraded surface. If, in any case, we fail to detect the point of inoculation, we are charged with having observed poorly. It is said also that the poison may have found lodgment by reason of an ozæna, abscess of the antrum, by means of a small scratch which heals before the disease manifests itself, etc.

Roger has recently cultivated the streptococcus of erysipelas, without the access of air, and finds that it develops toxic products which he was able to precipitate by means of alcohol, and which were destroyed by heat. The injection into the veins of filtered cultivations, *i. e.*, toxins, resulted in the development of a persistent predisposition to infection. The use of similar solutions, if heated to 250° F., conferred immunity.

Impaired nutrition, whether due to little or poor food, a lack of proper hygienic surroundings, general debility, Bright's disease, or chronic alcoholism, is the especial predisposing cause. Exposure to cold and damp seems occasionally to be a prominent etiological factor.

**Pathology and Morbid Anatomy.**—The tissue changes are those of simple inflammation, the intensity varying in different cases from an erythematous blush to the intense phlegmonous changes. Perhaps the most marked feature of the process is œdema, the fluid occupying the subcutaneous tissue spaces. After death this is often the only prominent pathological element present. It is, of course, more pronounced in the loose connective tissues about the eyes and in dependent portions. Suppuration and even gangrene occurs if the inflammatory process is sufficiently intense. As these conditions develop, the skin is apt to become paler, sometimes of a livid appearance, pits readily on pressure, and is ultimately blackish and crackling. The appearance of vesicles upon the inflamed surface is quite common, and in rare instances they may be transformed into illy-developed pustules. The erysipelatous inflammation is in most instances external to the fascia. But occasionally deeper tissues and organs are involved. The various serous membranes, *viz.*, peritoneum, pleura, pericardium, and meninges are sometimes attacked. The inflammatory process may creep into the external auditory meatus, and involve the deeper structures of the ear; or into the nose and mouth, involving the pharynx and larynx, or *vice versa*.

The lymphatic glands connected with the inflamed area are frequently swollen, sometimes prior to the appearance of the inflammatory process. The lymphatic vessels leading to the swollen glands may be perceptible as red streaks upon the skin.

There are no special blood-changes. Those which exist resemble the alterations found in the infectious fevers generally. The spleen, liver and kidneys are congested and softened. In certain of the fatal cases in which the cutaneous lesions were of mild degree, no evidence of the latter is found at the autopsy.

**Clinical Course.**—The incubation period ranges from two or three

days to one week. The symptoms of erysipelas are due to the local cutaneous process, to inflammatory fever, and to the systemic poisoning by the specific germ, and, occasionally, to complicating lesions. As erysipelas of the face and scalp is by far the most frequent form which the physician is called upon to treat, I will first describe this form and then point out the prominent features of the less common varieties. The symptoms of course vary in their intensity with the extent and severity of the local lesion. The period of invasion is generally characterized by a well-marked chill or chilliness, followed by a rapidly developing fever. At once or within twenty-four hours, a patch of inflammation appears at some point upon the face or scalp, far more frequently upon the former, however. This inflammatory change may occur at the site of a small ulcer or fissure or some other form of tissue-injury. Again, there may be an entire absence of any visible local condition which can be considered as determining the development of inflammation at the affected point. The nose is oftenest the point of beginning, some portion of the cheeks the next most frequent focus. The visible tissue-changes are redness, swelling, tension, sheen, œdema, and vesiculation in most cases; and these phenomena are attended subjectively by pain, burning, and stinging. The inflammation often spreads visibly from hour to hour, until the greater portion of the integument of the head is involved. In exceptional cases, the disease extends to the neck and even to the body.

With the progress of the local lesion, there is increase of the temperature and pulse, slight chills, pains in the head, back and extremities, anorexia, coated tongue, and perhaps nausea and vomiting, constipation, scanty urine, etc. The temperature seldom exceeds  $104^{\circ}$  to  $105^{\circ}$  to  $105.5^{\circ}$  F., although  $106^{\circ}$  F. or more is occasionally noted. The daily exacerbation of the fever usually, though not necessarily, occurs in the evening.

Many cases are attended by a high degree of prostration. If the attack is of an aggravated character, symptoms of a typhoid nature supervene, or cerebral symptoms become prominent. The latter class of symptoms may be due to actual inflammatory changes in the skull, but more frequently, as DaCosta has pointed out, to the toxic influence of the specific agent upon the cerebral tissues. In a small percentage of cases of erysipelas, the cutaneous inflammation, regardless of its point of origin, may spread over large tracts of skin, or, as I have several times seen, even of the entire surface of the body. Fortunately, as the inflammation spreads forward, it diminishes in the rear, so that the area involved at one time is not often very great. In these cases of migrating erysipelas the temperature is liable to attain and maintain a high point. In one case treated by me some years since, the temperature fluctuated between  $103.7^{\circ}$  F. and  $105.3^{\circ}$  F. for over four weeks, yet recovery finally ensued. Almost every square inch of the surface of the body was involved at one time or another during these four weeks.

Typhoid symptoms are especially apt to occur in such protracted cases.

It occasionally happens that in the course of facial erysipelas the eyes become involved in the inflammatory process. This is always a serious complication, and may lead to impairment of vision, if not destruction of the eyes.

Slight delirium is not uncommon in all varieties. The "mind wanders," the patient imagines much. Slight mental aberrations may pass on to higher grades, even unto maniacal raving. Coma supervenes prior to death in most of the fatal cases. In the extremity of many severe cases involuntary diarrhœa may occur. Albuminuria is very common in cases occurring in patients past middle life.

Classifications of the disease are artificial and of very little practical value. The merest tyro can see that they merge one into the other. In one case even, the observer may find one part highly œdematous, another smooth and shining, a third covered with vesicles, and still another gangrenous, and these parts merge into one another by almost insensible gradations. The term *simple erysipelas* suggests involvement of the skin only; *phlegmonous erysipelas*, that the subcutaneous cellular tissue is also involved. The terms *gangrenous*, *œdematous*, *suppurative*, etc., as applied to this affection, are sufficiently indicative of their meaning. When the disease becomes phlegmonous the resulting pus may accumulate at one or more foci, or be diffused, giving to the surface a characteristic boggy sensation. Gangrenous parts become livid or black and crackling, and are often the seat of intense burning pain. The various internal inflammations due to this poison present much the same symptoms as when occurring from other causes. The following have been especially noted: Meningitis, septicæmia, ulcerative endocarditis, pneumonia, pericarditis, and peritonitis.

**Sequelæ.**—The sequelæ of erysipelas are not numerous. The most important condition remaining, appears to be one of increased susceptibility to the disease, as one attack is a common forerunner of another. The skin involved is sometimes left in an indurated condition, especially the skin of the nose, and eyelids. Sometimes a very marked degree of thickening of the skin is a prominent result.

Abnormalities in sensation may result from changes in the nerves of the skin. If the scalp is affected, the hair may fall, but the loss is not permanent. Small abscesses may form subsequent to the erysipelas. Troublesome neuralgias have been observed. Recurring erysipelas is often due to chronic diseases of the skin, such as eczema. Irritated warts, or moles, fistules, or chronic ulcers, may constitute points of fresh infection.

**Diagnosis.**—Erysipelas presents such characteristic features that when present, it hardly ever escapes recognition. Diagnostic errors do



arise, however, respecting this disease, but nearly all of them relate to conditions like dermatitis, eczema, erythema, pemphigus, urticaria, and the lesion of rhus poisoning, which are carelessly diagnosed as erysipelas by either the laity or the profession. Of these, dermatitis probably affords the most excusable source of error. This latter condition is not associated with the marked febrile reaction of erysipelas, nor has it the peculiar shiny rose-red hue of the skin; and the constitutional symptoms of erysipelas are absent.

**Prognosis.**—Erysipelas of the head, as seen in my practice, has seemed to be a more serious disease than the books generally indicate. While none of my cases have yet proven fatal, many have presented most grave symptoms, and have had periods during which there was great doubt as to the result. This opinion has been strengthened by the knowledge of several fatal cases in the care of admirable clinicians. Death is brought about by quite a variety of conditions, *e. g.*, cerebral poisoning, or inflammation, exhaustion from protracted high temperature, suppuration, gangrene, or a simple protraction of the disease in a deteriorated constitution. In all persons past middle life, as well as in all possessing unsound vital organs, the risk is great. Recurring erysipelas is apt to affect persons suffering from chronic debility, or seriously impaired nutrition from any cause. I have treated one feeble old lady upon nine different occasions within ten years for a typical erysipelas of the face and scalp.

**Treatment.**—The first precaution is the isolation of the patient, lest the disease be carried to others. Rest in bed is absolutely necessary. The strength of the patient must be preserved. Accordingly a nourishing liquid diet with plenty of water to drink must be administered. Cool sponging of the surface should be practised if the temperature is high. Constant use of water dressings from the first, is an important adjuvant.

During his attendance upon a case of erysipelas the physician should not visit parturient patients, or those having open wounds, lest he spread the infection.

Of the various remedies recommended, *graphites* has seemed to be the most generally efficient. The writer has enough confidence in it to administer it first if there are not clear indications for another medicine. The third to the sixth trituration in doses of one grain every one to three hours, is recommended. The nature of the remedy may lead some, as it did me, to avoid it in so serious a disease. The presence or absence of vesiculation, the side and part affected, and other of the ordinary indications, seem immaterial.

In the ordinary smooth variety with active inflammatory changes, *belladonna* is of undoubted value.

*Apis* is to be preferred if œdema is marked, or the inflamed surface

pale. Apis proved curative in a case of erysipelas of the throat with œdema of the larynx and of the tongue.

*Rhus tox.* is indicated in vesicular erysipelas, especially if typhoid symptoms are present.

*Arsenicum* follows *rhus tox.* well in the typhoid variety, and was recommended by Jahr for migrating erysipelas.

For the cerebral symptoms, *lachesis* and *cuprum* have proven of most value; the former for the cerebral intoxication, and the latter for the inflammatory complications.

In protracted migrating erysipelas, *sulphur*.

In the phlegmonous variety, if *rhus tox.* is of no avail, *hepar, sulphur*, or *mercury*, is often indicated and may render good service.

For suppuration, *hepar, mercury*.

For gangrene, *carbo veg., arsenicum, lachesis, secale*.

For protracted suppuration and prostration, *arsenicum, cinchona, silica*.

*Aconite* is often useful in the early stages of phlegmonous erysipelas. It frequently diminishes the inflammatory symptoms and modifies the further course of the disease, even if it does not abort the attack, as some claim it will occasionally do.

*Veratrum viride* is considered by Hughes as preferable to *aconite* in the early stages of phlegmonous erysipelas.

*Stramonium* has controlled the cerebral symptoms when the characteristic delirium of the remedy has been present. The urinary symptoms of this medicine likewise aid in its selection.

*Borax, euphorbia, hydrastis, camphor, cantharis, croton tig., ipecac., kali carb., phosphoric acid*, and *ruta*, have also been recommended.

Incisions may sometimes be necessary to relieve tension and provide exit for the escape of pus and sloughs.

Many and varied have been the suggestions respecting the local treatment of erysipelas. Their value still remains very doubtful. Many of them are without doubt positively harmful, and have been justly condemned by physicians of all schools. Some of them serve merely to amuse the patient and his attendants, convincing them that active measures for relief are being taken. Cold-water dressings have already been mentioned. To be efficient, they should be changed frequently. Another application that is permissible, is rye flour. This is open to the objection that it obscures the appearance of the inflamed parts.

*Ichthyol* has in my practice proven useful. Its combination with flexible collodion has been the customary mode of application.

## SEPTICÆMIA AND PYÆMIA.

Both of these processes occur most frequently in connection with surgical practice; but inasmuch as they are occasionally observed in cases apparently medical, their consideration in this work is appropriate. The medical causes of septicæmia and pyæmia have been clearly stated by Roberts\* as follows: “(1) Diseases of bones, either acute or chronic, leading to suppuration; it may thus arise from disease of the temporal bone. (2) Affections of the heart or vessels engendering septic materials which contaminate the blood, as for example, ulcerative endocarditis; softening of clots, especially in the veins; phlebitis. (3) Abscess or gangrene in any part, either external to or within the organs. (4) Ulceration of mucous surfaces, such as that of the gall-bladder or its duct, or of the intestines. (5) Inflammation of a low type and attended with suppuration, implicating the pelvis of the kidney, the bladder or the urinary passages. (6) Diseases characterized by external inflammations of an unhealthy character, leading to the formation of pus, especially certain varieties of erysipelas, variola, vaccinia, in connection with revaccination, malignant pustule, glanders, carbuncles, or boils. Under this class may also be mentioned dissecting wounds and those from post-mortem examinations. (7) Low fevers occasionally, such as typhus, there being no evident local source of blood-poisoning. (8) Idiopathic pyæmia has been described, but it must be borne in mind that pyæmia may follow very slight injuries in unhealthy patients, and that there are many internal causes which may escape detection.”

### SEPTICÆMIA.

**Definition.**—A condition of the blood dependent upon the absorption of toxic matter produced by certain bacteria, principally those of suppuration. Clinically, it is characterized by fever associated with general asthenic symptoms; and pathologically, by the absence of foci of suppuration.

Cases of septicæmia may be divided into three classes, which, after all, represent rather differences in degree than variations in type. The mildest of these is generally referred to as reactionary fever. It is to this that the temporary rises of temperature succeeding surgical operations, traumatism, etc., are due. The fever may reach exceptionally 103° F., but it is not accompanied by any chill. If constitutional disturbances

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\* “Practice of Medicine.” Eighth Edition.



are present, they are very slight. The duration of the symptoms is variable, though usually but short. This condition was at one time regarded as due to reflex causes.

The second form of septicæmia is called sapræmia. It is dependent upon the absorption of the products of the bacteria of putrefaction. This is a far more severe type than the preceding, the intensity of the symptoms depending upon the quantity of morbid matter absorbed. It is ushered in by a rise of temperature to 103° or 104° F. in twenty-four hours or longer, after the operation or injury. The pulse is rapid. Constitutional symptoms are prominent, and consist of varying degrees of prostration, headache, delirium, restlessness, etc. The tongue is dry and glazed. The prerequisites for this form of septicæmia are the presence of dead tissue or a blood-clot in the wound, the infection of this wound by putrefactive bacteria, and the absorption of the ptomaines produced by the latter.

The third and most severe type is caused by the absorption of ptomaines from the local lesion, as in the last-described form; and in addition by ptomaines produced in the circulation by absorbed bacteria. The imbibed micro-organisms may reach the general circulation either through the blood or lymph vessels. The symptoms are of a far more severe type than the preceding. They may begin within twenty-four hours after infection, and are never delayed beyond the third or fourth day. They are ushered in by chill and fever, associated with profound constitutional disturbance. The fever is characterized by marked remissions and exacerbations of irregular occurrence, though nightly aggravation is the rule. The constitutional symptoms of sapræmia are present in an intensified degree. There is diarrhœa. The infected wound is red and swollen, in fact these local appearances may be widespread, and attended by lymphangitis and lymphadenitis. The patient finally sinks into a typhoid state, as manifested by feeble pulse, delirium, dry brown tongue, sordes on the tongue and teeth, and gradual failure of the circulation.

### PYÆMIA.

**Definition.**—A condition caused by poisoning of the blood by the bacteria of putrefaction, from a focus of suppuration within the body; and characterized clinically by recurrent chills, fever, sweat, profound prostration, and various associated symptoms dependent upon the organ or organs involved in the secondary suppurative process. Pathologically, it is characterized by the formation of multiple metastatic abscesses. While this disease is of peculiar interest to the surgeon, it is not without attraction to the physician on account of its general character, and for the further reason that it appears at times secondarily to slight lesions, such as ulceration of the mucous surfaces, which are treated

by the general practitioner. It is rarely independent of any apparent local cause (*idiopathic pyæmia*). The meaning of the term pyæmia (pus in the blood) suggests the older view concerning its pathology, *i. e.*, that pus was actually taken up by the vessels at the site of original lesions, and deposited at various points, developing thus purulent foci. It was also supposed that inflammation of the veins at the infective focus played a most important part.

**Pathology and Morbid Anatomy.**—The primary pathological cause of pyæmia is the absorption of the micro-organisms of suppuration arising from an accumulation or retention of pus in a part. These are the streptococcus pyogenes and the staphylococcus pyogenes aureus and albus. The former is the most frequently found. They exist both in the primary seat of suppuration and in the metastatic abscesses. The production of pyæmia by them is readily explained by the production of a coagulation necrosis in the smaller vessels about the suppurating part. Thrombi are thus formed, and are washed into the general circulation. In this manner they form centres of suppuration at their points of lodgment.

It is but a few years since pyæmia was a common sequence of surgical operations, but following the introduction of the antiseptic method of treatment, it has been very unusual. Our cases of pyæmia to-day are generally such as develop in connection with lesions which cannot be treated antiseptically, or but imperfectly so.

The seat of the primary suppuration may be at any point in the body. Usually, it is in the subcutaneous cellular tissue or in the pelvic cellular tissue and organs, or sub-peritoneal cellular tissue. It may be, however, in any of the organs, in the marrow of the bones, or in the middle ear. Pyæmia sometimes arises from ulcerative lesions of the cardiac valves, the so-called malignant endocarditis. Cases are said to arise in which no primary focus of suppuration can be found. Such statements are probably the result of deficient observation.

The sites of the secondary foci of suppuration will vary according to the location of the primary lesion. If the latter be in the subcutaneous cellular tissue or in the long bones, the metastatic abscesses appear in the lungs, the bases of which are most frequently attacked, presenting wedge-shaped areas of lobular pneumonia. If the emboli should pass through these organs without lodgment, the heart and kidneys are especially liable to suffer. If the primary disease be in the parts tributary to the portal veins, the secondary abscess will be in the liver. Following malignant endocarditis, the secondary abscesses may exist in any of the organs. The emboli may not all be septic, or the local condition may not be favorable to suppuration, which would account for some of the infarctions not passing to the stage of suppuration. As already indicated, the lungs are by far the most frequently involved. In very

acute fatal cases, the lesions may be confined to these organs. Acute pleurisy, either serous or purulent, may attend. This pulmonary localization may succeed external wounds, diffuse periostitis, osteo-myelitis, etc. Metastatic abscesses within the liver may arise from suppurative lesions anywhere within the domain of the portal circulation.

Idiopathic pyæmia has been referred to. Dr. Wilkes, especially, has called attention to a form secondary to endocardial inflammation (arterial pyæmia), in which large numbers of infective particles are thrown off from the vegetations in the left heart. The endocardial inflammation is apt to arise in the course of infectious diseases, or may be secondary to a suppurative focus in some portion of the body.

**Symptomatology.**—Owing to the multiplicity of points for the formation of abscesses, the great variations in the symptomatology presented by different cases of pyæmia can be well understood. The characteristic signs are those of suppuration, namely chill, fever, and sweats. These are associated with symptoms varying according to the organ or organs involved. The initial chill is usually a severe one. Still it may consist of merely a feeling of coldness. With this is a rapid rise of temperature. The fever is of a very irregular type. It may disappear entirely, only to come on again and reach an even higher point than before. It usually presents marked remissions in the mornings and rises again in the evening. The variations in individual cases may be as great as three or four degrees. The sweating may be either continuous or recur in paroxysms. The recurrence of chills is probably indicative of the formation of new suppurative foci.

To these general symptoms may be added a variety of local disturbances, the character of which depends upon the organs most affected; if the lungs, dyspnoea, cough, pain and the various signs of bronchitis, pulmonary consolidation, or pleurisy. To these may be superadded those of pericarditis. Peritonitis is not uncommon. The spleen is usually perceptibly enlarged and the seat of pain, which is due to perisplenitis. In rapid cases (five to ten days duration) stupor, delirium, a dry, brown tongue, a feeble quick pulse and other symptoms of the typhoid state become prominent. If the liver is involved there is usually some degree of jaundice; albuminuria, granular casts, pus and blood may likewise be present; if the intestines, diarrhoea may be pronounced. Symptoms referable to the presence of metastatic abscesses in the various organs and tissues not above enumerated will suggest themselves.

**CHRONIC FORM.** Pyæmia may pursue a chronic course. In these cases the viscera may escape, and the joints and superficial structures be attacked. The points of formation of abscesses are indicated by tenderness, redness, and the other well-known indications. Fresh abscesses continue to appear for several months. The patient ultimately recovers with impaired joints or dies from exhaustion. The symptoms of any



case, it must be remembered, depend largely upon the localization of the secondary lesions.

**Diagnosis.**—Typical pyæmia developing subsequent to the conditions which are known to so frequently precede it, presents no difficulties in diagnosis, but as it appears in association with otitis, lesions in the intestinal or genito-urinary tracts, or disease of the endocardium, it is frequently not recognized. In some cases, too, it may be confounded with early phthisis, Hodgkin's disease, impacted gall-stones, etc. The possibilities, however, favor the confusion of septicæmia and pyæmia with malarial and other low forms of fever. So far as intermittent fever is concerned, the typically regular recurrence of the chills and fever are sufficiently diagnostic. If more is needed, the rapid disappearance of all symptoms under the administration of quinine is conclusive. Typhoid fever presents some points of similarity; but the modes of onset of the two diseases are very different. The history of the cause of the trouble is an important diagnostic aid.

**Prognosis and Treatment.**—The prognosis in both septicæmia and pyæmia is very grave indeed. Severe cases are hopeless unless the original seat of infection and the metastatic abscesses are accessible to surgical interference. In the latter case, thorough cleansing of the point of infection on antiseptic principles, and complete evacuation and washing out of all abscesses, offer some hope of success.

When such treatment is impossible or impracticable, the attendant must satisfy himself with ministering to the comfort of the patient, conducting his treatment on symptomatic indications. The strength must be preserved as far as possible by stimulants and nutritious and easily digested food. Pain must be combated.

The remedies best adapted to these cases are *arsenicum*, *cinchona*, *hepar*, *rhus tox.*, *phosphorus* and *quinine*. *Arseniate of quinine* is an especially important remedy. *Cinchona* is called for by the intense prostration and the profuse sweats. Typhoid symptoms indicate *rhus* and *phosphorus*.

## SCARLET FEVER.

**Nomenclature.**—Scarlatina, scarlet rash.

**Definition.**—A specific contagious fever affecting mostly children, and characterized by the presence of a rash and sore throat, and very frequently followed by otitis, nephritis, rheumatism, and other sequelæ.

**History.**—There can be no question that scarlet fever is an ancient disease; still there are no clear accounts of it prior to the seventeenth century. At one time it was looked upon as a variety of measles, but differing from the common form mainly in that anginose symptoms replaced the catarrhal inflammations of the respiratory tract. The discrimination between the two diseases was first clearly made by Sydenham in 1661. He, however, saw or rather described only typical cases free from complications. His description of this type is classical, and has led to the designation “the Sydenham type of scarlatina” as applied to the uncomplicated disease. The first appearance of the disease in America was at Boston and Kingston in 1735. New York and Philadelphia were not infected until eleven years later. The appearance of the disease in the Western States is of comparatively recent date. Scarlet fever does not seem to have gained much of a foothold in Asiatic countries. Cases have occurred in Japan, but the susceptibility of the people seems to be so small, that epidemics have never occurred.

**Etiology.**—Scarlet fever is caused by a contagium transferred from the sick to the well by the atmosphere, by the medium of fomites, as well as by immediate contact with those suffering from the disease. As in the other infectious diseases, the *contagium vivum* is supposed to be a low form of vegetable life; but as yet a specific microbe has not been discovered. There are some who advocate the possibility of a spontaneous development of the disorder. Beyond all doubt, cases alleged to so arise have probably been exposed in some unknown manner. One attack of the disease generally protects from subsequent ones. Still, second and even third attacks are not unknown.

While there is abundant evidence showing that the specific element causing scarlatina is suspended in the air, and wafted about by atmospheric currents, there is strong evidence to the effect that the disease is more frequently conveyed by fomites, and that the atmospheric portability of the contagium is not so great as was at one time supposed. Articles of clothing, letters, and the domestic animals, have been carriers of the disease germs. Cases are on record where children were infected by their nurse taking from a trunk and wearing an old dress which she had

worn while in attendance upon a scarlatina case eighteen months before. Letters written in the sick-chamber have conveyed the disease to a distant household. Books taken from public libraries form an element of danger.

Quite a number of epidemics have originated from infected milk. The milk may become a bearer of the disease germs either by the existence of the disease in the dairyman's household, or by infection from the cow herself. The importance of the former method of milk infection has only been recently recognized. The existence of scarlet fever in the cow has been denied by some. Hill, however, reports an epidemic arising from bad milk supply. Investigation showed that one of the cows had a peculiar ulceration of the teats, and later desquamated very freely and became emaciated. That the animal's ailment was scarlet fever was positively denied by some. On abandoning the old milk supply, the epidemic disappeared.

The above views respecting the transmission of the disease obtain strong confirmation in the fact that it is generally prevented from spreading by careful isolation of the patient. In this respect, scarlet fever stands in sharp contrast to measles, for when the latter has once entered a household, it is seldom that any member thereof, unprotected by a previous attack, escapes.

Scarlet fever can be transmitted from one individual to another by inoculation. This fact has suggested the possibility of stamping out the disease by protective inoculations. Experience has demonstrated, however, that the disease thus artificially induced is even more severe than that of ordinary origin. Strickler, of Orange, N. J., has proposed inoculation of children with mucus taken from the domestic animals suffering from scarlatina. He has carried his idea into practice, and thus "vaccinated" a number of children with, he claims, the result of securing immunity. These experiments must remain for the present as without value, for it cannot be positively stated that the animals furnishing the virus really had scarlatina, or that the "vaccinated" subjects were really protected.

The tenacity with which the virus of scarlet fever clings to objects with which it has come in contact, is very great. Instances have been reported in which contagion followed the occupancy of a bed-chamber which had once harbored a scarlatina patient, and which had been closed for months. Personally, I have known a number of similar instances of contagion, and believe that municipal authorities should see that a house vacated by a family one of whose members has suffered from scarlatina or diphtheria, is thoroughly disinfected, or at least that the family subsequently moving into the house should not be made to face the contagion unwarned.

It is necessary in order that any substance become the medium for the



transmission of the poison of scarlet fever, it should remain in somewhat prolonged contact with the virus. This is well illustrated by the infrequency with which physicians and others who make but short calls on the sick communicate the disease to others. This fact, however, should not lead to carelessness in isolation; for cases are now and then observed in which a momentary exposure has resulted in infection.

The period of the disease at which there is the greatest danger of transmission is uncertain. Some claim that it is during the stage of desquamation, others that it is during the height of the eruption. The *London Lancet* Investigation Committee regarded the disease as contagious from the very first appearance of the eruption until the last vestige of cutaneous roughness had fled. Cases are known in which the disease has been communicated as late as the seventh week. The carriers of the contagium are the epidermic scales, the expired air, the mucous secretions, and the urine and fæces. The throat is believed to be a possible source of danger for some time after apparently completed recovery. Under such circumstances scarlet fever has been communicated by kissing. The prolongation of the course by various complications and sequelæ renders the danger period correspondingly long. Even such a condition as dropsy carries with it some danger to others.

The stage of incubation remains unsettled. It is believed to range ordinarily from three to six days. Development of the disease within six hours after exposure has been reported. In other cases, the appearance of symptoms has been delayed for weeks. Patients having open wounds seem to develop scarlet fever much earlier after infection than do other subjects.

Age is a powerful predisposing factor in the etiology of scarlet fever. The disease may attack subjects of any age, but children are the most susceptible to it. The greatest liability appears to be at the age of three or four years. Each year after that lessens individual susceptibility. The severity of the disease is greatest during the first two years of life.

Scarlet fever is endemic in certain sections, especially in the larger cities and towns, a few cases being always about, particularly in the winter months. Such cases must always be regarded as the result of contagion, and not as evidence that scarlatina may arise *de novo*. Under certain circumstances with which we are not at all well acquainted, the disease becomes epidemic. We do know that some sort of intercourse with the sick is necessary for the transmission. The type and intensity of the fever present great variations in different seasons and during certain epidemics. Without assignable reasons, scarlet fever which has existed in a mild form in a community for years, suddenly puts on a new physiognomy, and assumes a malignant character. Even the symptomatology varies with the epidemic. At one time, certain features are prominent, at another, others. Thus in some epidemics the throat symptoms

attract the most attention, indeed "diphtheria" may appear to be a common complication. At other times, post-scarlatinal nephritis is the rule. I think from what I have read, that scarlet fever must be upon the whole of a more serious type in England than on this continent.

The origin and nature of surgical scarlet fever have been extensively discussed. The existence of this condition has been denied by many, who believe that the phenomena of a septic fever have been misinterpreted. Surgical scarlatina has, notwithstanding this, many good authorities who support it. None of them, however, have offered any very good reasons for its *raison d'être*. Hutchinson has suggested that it is the result of the prolonged survival of quiescent germs, the activity of which is favored by the disturbance produced by the operation. Others believe that the open wound offers unusual facilities for the absorption of the virus.

Pregnancy acts as a predisposing factor, though not as much so as the lying-in and lactation periods.

**Pathology and Morbid Anatomy.**—Aside from the skin changes, there are no characteristic anatomical lesions distinguishing scarlet fever.

**SKIN.** The eruption makes its appearance usually within twenty-four hours after the febrile onset. There appear upon the skin numerous circular points, slightly elevated and so closely aggregated as to give the cutaneous surface a diffused redness, which has been compared in color to that of a "boiled lobster." In slight cases the spots remain discrete, the skin between the points of eruption remaining quite or nearly normal. In the aggravated forms of the disease, there is a general turgescence, in some cases even a positive erythema of the skin, with marked swelling, glossiness, heat, itching, etc. In certain cases, owing to the intensity of the cutaneous inflammation and the high degree of change in the blood, petechiæ and even extensive ecchymoses appear.

**THROAT.** The throat changes consist of an inflammation of the tonsils and adjacent connective tissue and lymphatics. The lymphatics at the angle of the jaw are generally swollen, and the neighboring structures œdematous. The swollen glands frequently suppurate. The greatest variations in the intensity of the throat inflammation are noticed. In mild cases there may be nothing more than a marked hyperæmia of the pharyngeal and nasal mucous membranes, with involvement of the palate and tonsils. Sometimes the latter are considerably enlarged. Abscesses may form in them or in portions of the mucous membranes. Again, the mucous membrane may be livid in color and highly œdematous. In severe cases the inflammation may extend to the deeper structures and give rise to extensive swellings and suppuration. Even gangrene and sloughing may ensue. Bourges, who has given the throat symptoms of scarlatina considerable study, divides them into the erythematous, pseudo-membranous and gangrenous. The pseudo-membranous variety has frequently been confounded with

diphtheria, and with good reason too, for there are no reliable data, aside from bacteriological examination, to differentiate these conditions. The pseudo-membranous anginas of scarlatina may be considered as of two kinds, according to their time of appearance, namely, early and late. The early variety has no relation whatever to diphtheria. The late cases are usually of diphtheritic origin. Bourges has divided the early cases into three classes, as follows: The *benign*, in which the membranous deposit is not extensive, and the lymphatic glands are but slightly, if at all, affected; the *grave*, in which there is rapid extension of the membrane, persistent glandular involvement, and general systemic disturbance; the *septic form*, in which, to use the author's own words, there is a characteristic picture of hypertoxic angina.

**EARS.** Extension of the inflammatory process in the throat to the ear results in suppuration in the latter, with destruction oftentimes of important structures. It is one of the most frequent of the so-called complications of scarlet fever.

**KIDNEYS.** It is most probable that hyperæmia of the kidneys exists in most cases of scarlet fever. This hyperæmia, as well as the subsequent acute parenchymatous nephritis (which develops in a large percentage of cases), should be looked upon as a prominent feature of the disease, rather than an actual complication. The tendency of the virus to attack the epithelium of the kidney is almost as great as the tendency to involve the throat. Some authors have claimed a catarrh of the kidneys to be invariably present.

**LIVER.** Occasionally the liver is found enlarged; the result, according to Klein, of an acute interstitial hepatitis.

**SPLEEN.** According to Klein, the spleen is larger than normal, is hyperæmic and shows enlargement of the Malpighian tufts and other minute changes.

Rarely are the brain and its envelopes congested. The alimentary tract is in a state of catarrhal inflammation, which may be followed by some exfoliation. There may be bronchitis (simple or capillary), pneumonia (catarrhal or croupous), and inflammation of any of the serous membranes.

**Clinical Course.**—The onset of a typical uncomplicated case of scarlet fever is usually sudden. If prodromata exist, the age of the patient prevents complaint, consequently they usually pass by unnoticed. Chilliness or a slight rigor is common; but, like the prodromic symptoms, it is often overlooked. Sometimes the disease is ushered in by a convulsion. The symptom which usually first attracts attention is the fever. The temperature rises very rapidly, and the increase in the pulse-rate is in direct proportion to the febrile rise. Headache, sore throat, vomiting, mental dulness, bodily restlessness and anxiety, attend the early rise of temperature. Delirium is sometimes observed, and prostration is pro-



found. The symptoms all increase in intensity from twenty-four to thirty-six hours, when the characteristic eruption appears. This first shows itself about the neck and chest. With the full development of the disease the tongue assumes a "strawberry" appearance, the buccal cavity becomes inflamed, and the lips, teeth and gums covered with sordes, the throat ulcerated, the lymphatics of the neck and behind the angle of the jaw enlarged, the urine scanty, high-colored, and perhaps albuminous. The increase of the color and extent of the eruption is rapid. At the close of the third or fourth day the trunk, face and portions of the extremities being fairly covered, *i. e.*, the second day after the first appearance of the rash. The rash reaching its height, there is a gradual decline in the eruption, it finally disappearing between the sixth and the ninth days. With this, the temperature declines, as do all other symptoms. Desquamation begins with the disappearance of the eruption. The temperature then becomes normal, unless there is some focus of inflammation, as in the throat, ears, lymphatics or kidneys.

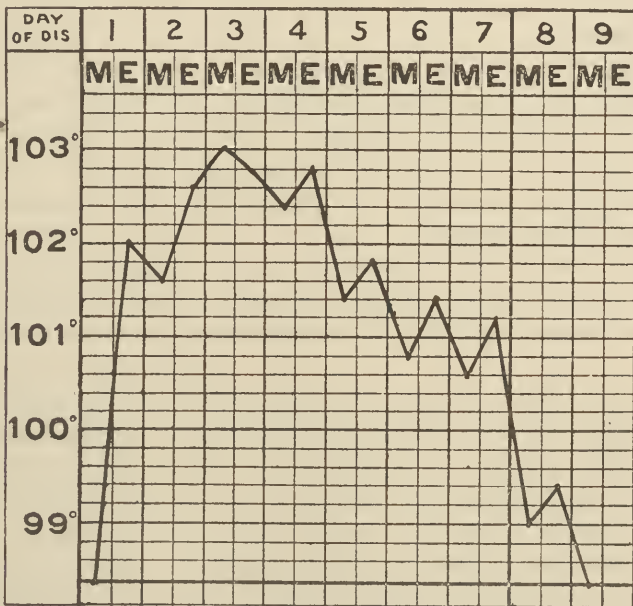


FIG. 9. TEMPERATURE CHART OF A CASE OF SCARLET FEVER.

**Analysis of the Symptoms.**—TEMPERATURE. Excepting in the mildest cases the rise in temperature is very rapid, a rise of five degrees in the course of the first twelve hours being quite common. This rise continues until the appearance of the eruption. The subsequent course of the fever varies greatly in different cases. If a typical case, that is, one of average intensity without marked complications, thermometric observations show slight daily remissions, until the fourth

or fifth day, at which time there is marked fall in the evening fever; and each succeeding day finds the morning and evening temperature dropping lower and lower, becoming normal with the disappearance of the eruption or early in the stage of desquamation. In the malignant form the initial rise is frequently to as high as  $105^{\circ}$  F., or even  $106^{\circ}$  F. or  $107^{\circ}$  F., within the first twenty-four or thirty-six hours. I have seen a rise of eight and one-half degrees within eight hours after the initial chill. Death took place in convulsions eleven hours after the appearance of the chill. However severe the fever, the temperature declines somewhat upon the proper development of the eruption, unless there is a high grade of inflammation in the throat, skin or other organs. In mild cases the rise in temperature is very slight, many cases running their entire course without the thermometer once registering over  $100^{\circ}$  F. Cases have been reported as occurring without fever; but the mild character of such has prevented observation, and possibly an early period of pyrexia has been overlooked. The skin is remarkably dry with this heat. Exceptionally, cases in which marked perspiration is the rule, will be observed.

A *secondary fever* has been described by Gumprecht. This occurs independently of any apparent complication. It usually sets in after the temperature has reached the normal. The author attributes it to a secondary infection. It does not render the prognosis of the case any more grave, but it serves as a warning to prepare for other complications later in the course of the disease.

Talamon has described cases in which the fever persisted without abatement until the entire disappearance of the rash. It may even last eighteen or twenty days, and it constitutes an element of considerable danger.

**PULSE.** The pulse keeps pace with the rise of temperature. In the malignant form, and in fact in any variety in which the temperature is high, the pulse may range from 140 to 180 per minute. It does not show such a marked tendency, however, to slow up with the subsidence of the fever, but remains rather rapid during convalescence.

**THE ERUPTION.** The eruption appears usually within the first twenty-four or thirty-six hours. Every hour that its appearance is delayed beyond that time adds to the seriousness of the case. In a small percentage of cases, however, the eruption may be delayed until the third day without any serious symptoms intervening. As already stated, general observations show that it appears first about the neck and upper portion of the trunk and face. Still there is no definite rule as to the point at which its first manifestations will be met. My own observations have led me to look first for it on the soft palate or roof of the mouth. The rash spreads rapidly to the extremities, reaching the stage of maximum development generally upon its second day. In rare cases, the full develop-

ment may be attained on the first, while in others it may be delayed until the third day. When appearing upon the extremities, it is especially apt to affect the flexures of joints. There is decidedly less tendency for the eruption to affect the face than other parts. It is hardly ever well developed over the forehead, temples or chin, and it spares the cutaneous surface for quite an area around the mouth. As first seen, the eruption consists of little fine reddish dots, which vary somewhat in size in different cases. In active cases, partly as the result of confluence of the eruptive points, and partly from the general hyperæmia of the skin, large portions of the body present a uniform redness. Such areas itch and burn. Even in cases of great intensity the rash does not always become confluent. It sometimes takes the punctate form, and is decidedly uneven in its distribution; in other words, it is patchy. In mild cases the eruption remains discrete; indeed it may then be mistaken for "prickly heat." Sometimes the eruption on the fingers is *papular*—this I have noticed in quite a number of cases.

The color of the rash presents some variations in individual cases. Characteristically it is a bright red, and has been aptly compared to the color of a boiled lobster. Attendant circumstances affect the intensity of this color somewhat. Thus when the surface is kept cool, the color pales; while if the child is kept inordinately warm by too heavy clothing or too close a room, the red will be of a brighter hue. In malignant cases the rash often assumes a purplish hue. Occasionally the eruptive points are observed mostly about the hair follicles, and are of a dark red color.

It is not necessary that the rash affect the entire surface. There are cases in which only portions are involved without the advent of any serious symptoms whatever.

The rash begins to disappear about the sixth day; occasionally it does so about the fourth, and is entirely gone by the ninth day. Usually it clears up first on those parts which were first invaded. Desquamation then follows. In general this takes place in very fine scales, but when the inflammation has been intense, large flakes of epidermis are cast off from the hands, feet, etc. These may even take the form of the part from which they came. Sometimes after desquamation has been apparently completed, the process is repeated again and again. A disappearance of the rash without at the same time a subsidence of the febrile and other symptoms is of unfavorable omen, usually portending serious cerebral complications.

THE THROAT. Sore throat is one of the earliest symptoms, and depends upon a catarrhal inflammation of the fauces. The inflammatory process frequently extends upward into the nasal cavities, and forward into the mouth. With the progress of the disease, the deeper structures of the throat are involved. The tonsils especially enlarge,



sometimes even to the extent observed in phlegmonous tonsillitis. They may even exhibit quite deep ulcerations, the surface of which may be covered by a mouldy looking exudate. The cervical lymphatics enlarge, the degree of enlargement usually being commensurate with the intensity of the changes in the throat. There is intense pain on swallowing, and, sometimes, regurgitation of food through the nostrils. The sore throat of scarlatina often bears a close relationship to diphtheritic angina. Indeed the nature of the membrane in the scarlatinal sore throat was long a matter of discussion. It is now positively known that a pseudo-membrane peculiar to this disease may appear, and that an actual diphtheritic inflammation of the throat may supervene, especially in the later stages of the disease. This subject has been already referred to when speaking of the pathology and morbid anatomy of scarlet fever.

There is hardly a symptom of scarlet fever which may present such wide variations in intensity as does the sore throat. It may be absent, or at most consist of but a slight redness. In extreme cases, the inflammation assumes a phlegmonous character, with extensive suppuration of the connective tissue substance about the neck.

EARS. Suppurative inflammation of the middle ear is a frequent complication of scarlet fever. It results from the extension of the inflammation from the throat to the ear by way of the Eustachian tubes. It usually appears towards the decline of the fever or the beginning of convalescence. The symptoms manifested do not differ materially from those characterizing otitis media due to other causes. Usually there is severe pain, followed in from one to three days by a perforation of the membrana tympani, and a discharge of pus from the external auditory meatus. This discharge of pus is usually followed by decided relief of pain. This latter is not always the case, however, for the reason that the middle ear inflammation continues unabated, or the opening in the drum-head is so small or so unfortunately placed as to give inadequate drainage; while in others, involvement of the mastoid cells keeps up the suffering. Until recent advances in the treatment of this complication, the post-scarlatinal otitis media often continued uncontrolled until destruction of this intricate organ resulted, followed by permanent impairment of hearing. There would often appear recurrent attacks of inflammation with purulent discharge, still further damaging the hearing. In some cases even extension of the suppuration to the brain occurs. This latter danger is not an imaginary one even in the acute stages of the disease. Prompt and intelligent treatment must be adopted in every case of "earache" accompanying scarlatina. It is wise to make regular systematic examinations of the ears throughout the course of the disease, for sometimes a suppurative otitis media sets in so insidiously as to do extensive damage before its symptoms force its recognition.

MOUTH. During the first day the tongue usually presents a slight

whitish coating, with red tip and edges. This coating exfoliates rapidly, leaving the tongue red, glazed, and sometimes raw and cracked. The most characteristic feature consists in the hyperæmic and enlarged papillæ constituting the "strawberry tongue," which is so generally regarded as strongly suggestive of scarlet fever. The mucous membrane lining the buccal cavity presents more or less the same appearance as the tongue. The lips are dry and cracked and the corners of the mouth often fissured.

**Nose.** The mucous membrane lining the nasal chambers is generally inflamed and secretes a thin discharge. The edges of the nostrils are sometimes dry, sore, and fissured. The upper lip may be irritated by the nasal discharge.

**GASTRO-INTESTINAL TRACT.** Vomiting is one of the early symptoms accompanying the initial fever. Indeed it is regarded by many as characteristic. Associated with the headache, sore throat, and high fever, it forms a quartette of symptoms of great diagnostic value. If the initial stage is passed without vomiting, that symptom may be looked for with the appearance of the eruption. The vomited matters are ejected suddenly and with considerable force. The appetite is impaired; thirst is great; the bowels may be either loose or constipated.

**KIDNEYS.** Acute nephritis has for a long time been considered a frequent accompaniment or sequela of scarlet fever. Recent investigations, however, by Klein and many others, indicate the existence of slight degrees of nephritis during the course of scarlet fever very much oftener than was at one time supposed. Indeed, some authors, including Friedrich, Begbie, Bönning, and Stevenson Thompson, teach that the evidences of nephritis are always present in fatal cases. Opposed to their views are the observations of Friedländer, who in 229 autopsies upon patients dying of scarlet fever, found evidences of nephritis in less than one-half. According to Eisenschütz and others, renal catarrh, as indicated by cylinders of renal epithelium, mucous casts, an increased amount of mucus in the urine, but an absence of albumin, is quite constant. Such a catarrh of the kidneys is not peculiar to scarlet fever, but occurs in connection with many of the acute infectious disorders. The frequency of nephritic manifestations varies greatly in different epidemics, the number affected in some being fully ten times as great as in other times of general prevalence of the disease. These notable disparities can only be accounted for by difference in the character or intensity of the scarlet fever virus. There is a tendency to look upon the dropsy as the first indication of kidney inflammation. This is a serious mistake, and leads to delay in the application of proper treatment. Fleischmann's series of 472 cases of scarlet fever with 95 cases of nephritis are interesting as showing the period at which dropsy arises. This condition occurred 9 times during the first week; 30 times in the second week; 23 times in the third; 20 times in the fourth; only 5 times subsequent to the fourth week.

Some of the most intense cases of post-scarlatinal nephritis are practically unattended by dropsy.

In view of the observations that indicate the great frequency of some degree of nephritis during the course of scarlet fever, it becomes the duty of the physician to make frequent urinary examinations in all stages of the disease, in order to detect the earliest evidence of the existence of kidney changes prior to the development of dropsy and the various conditions of the urine which are sufficiently obtrusive to attract the attention of even the laity. Slight albuminuria, the principal evidence of the disease, is sometimes discoverable a week or more before the dropsy appears. All cases of scarlet fever, in which convulsions, coma and other evidences of cerebral disturbance set in after the full establishment of the eruption, find in the kidneys a possible cause for the complications. Many cases of so-called malignant scarlet fever have been cases in which the essential feature was really uræmia. The urine in scarlatinous nephritis presents various quantities of albumin, blood and tube casts—the latter being especially of the blood and granular epithelial type—and is diminished in quantity, or even entirely suppressed. In typical cases the patient passes a small quantity of urine which deposits a dark brownish or reddish sediment. Dropsy is usually first manifested in the face about the eyes, but soon becomes general. In the higher grades of nephritis effusion may take place into any of the serous cavities, and may even be associated with inflammation of the membranes lining the same. This effusion may be sufficiently great to produce alarming symptoms, if not death, by its mechanical effects. Vomiting is a frequent symptom in scarlatinal nephritis. I have observed two cases in which the persistence of this symptom during the course of light attacks of the disease was accounted for by the discovery of albuminous urine and other evidences of slight nephritis. In neither of these cases was the fever, dropsy or any visible evidence in the urine suggestive of the nature of the trouble.

Uræmic manifestations in the shape of convulsions, amblyopia, coma, delirium, or headache, have been known to suddenly make their appearance when the existence of nephritis had not even been suspected. The convulsions may present most irregular types. Very frequently the patient goes from one into another in rapid succession, even until death closes the scene.

The period at which scarlatinal patients may be regarded as freed from the danger of nephritis has been variously estimated. It is generally held at the present time that this complication does not develop after the seventh week. The duration of the trouble is likewise variously stated. Most cases are mild, lasting but a few days or weeks. Still there are many in which the albuminuria persists for several months. The best clinicians now believe that the unrecognized acute nephritis of scarlet



fever is responsible for much of the chronic Bright's disease in young people. It may well be questioned if it is not a frequent cause of kidney trouble in later life.

A dropsy independent of evidence of kidney disease has been described, and variously attributed to changes in the connective tissues and to anæmia. Such interpretation of this symptom must be received with a great deal of caution, for serious, even fatal, nephritis has occurred without the advent of albuminuria.

**NERVOUS PHENOMENA.** In the milder forms of scarlet fever the nervous disturbances are slight. There is early some headache and general pain. The patient is perhaps restless, or may be drowsy and quiet. In quite young subjects, the attack may be ushered in by convulsions, not necessarily associated with a high temperature; but more frequently, perhaps, convulsions occur with the advent of the eruption. Occurring at this time, the symptom need not excite alarm. If convulsions develop later, their dependence upon nephritis should be considered. At this time, whether due to nephritis, hyperpyrexia, or toxæmia, they are of grave import. In the malignant form of scarlet fever, or one of the malignant forms—for there are varieties manifesting malignancy—coma may supervene without a degree of fever that may be considered hyperpyretic. Early comatose cases are the most hopeless of all the malignant forms; many such cases being instances of a coexisting meningitis.

**Complications and Sequelæ.**—The otitis and the nephritis already described are ordinarily regarded as complications or sequelæ of scarlet fever. In addition to these we have quite a variety of others, of decidedly less frequency, however. Rare, though none the less interesting, is the simultaneous occurrence of scarlet fever and one of the other exanthemata. Thus we may find scarlet fever associated with smallpox, varicella, measles, typhoid fever, or whooping cough. Conversely one of these diseases may be the primary affection and scarlatina may be added to it. Diseases of the respiratory mucous membrane and acute lobar pneumonia have been observed. Inflammatory affections of the serous membranes, as pleuritis and peritonitis, are somewhat more common.

Of far greater frequency than the above are the ulcerative and suppurative complications. These may affect any part of the body. Thus there have been observed parotitis with suppuration, ulcerations of joints and genitalia, extensive ulcerations about the throat leading to vascular erosions and hæmorrhage, and epiglottidean abscess. Even multiple gangrene has been reported.

The joint complications are tolerably frequent. They are generally referred to in literature, though incorrectly, as post-scarlatinal rheumatism. They should be called properly, "synovitis." This affection usually runs a mild course, and may affect almost any of the larger joints. It may end in suppuration and permanent deformity of the joint. As a

rule, it is not associated with heart troubles. There is a form of synovitis which occurs at about the third or fourth week of the disease, especially in young children, which is frequently associated with endocarditis. These joint affections are all believed to depend upon a special infection by a streptococcus.

Nervous sequelæ and complications have been assigned to scarlet fever. They include, prominently, chorea and epilepsy. Less frequently paralytic affections are due to organic changes consequent upon the altered blood state.

**Malignant Scarlatina.**—It has been stated that scarlet fever of malignant type exists in several varieties. Thus we recognize a type in which the patient succumbs within a few hours of the onset, overwhelmed by the intensity of the poison. In such cases symptoms have scarcely time to develop, and the diagnosis is made by the knowledge of epidemic influences. There is a *hæmorrhagic* form in which epistaxis, hæmaturia, and petechiæ are prominent. Lastly there is the *anginose* variety in which throat symptoms are of a most malignant type. These latter have already been referred to.

**Relapses.**—Relapses are possible, though rare. They come on in from six to ten days after the disappearance of the original attack. Most authorities regard them as of less severity than the first seizure.

**Diagnosis.**—The diagnosis of scarlet fever is usually made with ease. The rapid onset, with febrile symptoms, associated with headache, sore throat, and vomiting, the prevalence of the disease, and the susceptibility of the patient, by reason of age and previous freedom from the disease, are all strongly suggestive. But the true nature of the affection is positively manifested on the appearance of the characteristic exanthem. The difficulties in diagnosis are encountered in the irregular forms of the disease. In the very mild cases we are obliged to wait for the appearance of the eruption, which may then be so illy-developed that we are not always certain of the nature of the affection, even then until desquamation occurs, or some complication, as nephritis or otitis, sets in. In the malignant form, in which coma and death occur early, that is within the first twenty-four or thirty-six hours, the eruption not having appeared, we are frequently obliged to make a diagnosis upon such evidence as the existence of an epidemic, or the presence of other cases in the same household.

**Prognosis.**—The mortality in scarlet fever is exceedingly variable. The character of the epidemic influences it more than in most diseases. Mortality rates differ widely, as from one in three, to less than one in a hundred, have been reported. Therefore one must always take into account the nature of the epidemic in making a prognosis and determining whether the treatment pursued has been relatively successful. According to Wm. Squire, the mortality in 3,984 cases treated in special

hospitals in London in three years (1877 to 1879) was 449. He also states the general mortality to be from 6 to 16 per cent. and that the percentage of deaths is not less in non-epidemic years. This is much too high a rate to represent the results from the homœopathic treatment of scarlatina. As regards my personal experience, I would say, that while I have not recorded my cases, that in a practice of over twenty years, embracing hundreds of cases, my deaths from scarlet fever have been only six—undoubtedly less than 2 per cent. of the whole number treated.

A regular course, even if the temperature is high, and an absence of severe nervous or throat symptoms, as well as of the later nephritis, joint affections, extensive lymphatic or cellular tissue swelling, warrants a favorable opinion as to recovery. While hyperpyrexia in the course of scarlet fever must not be underestimated as to its serious influences, yet it must not be overlooked that scarlet fever is a disease in which in typical cases the pyrexia is always high, the temperature mounting to a height which in many other febrile affections would indicate the progress of the disease to be most unfavorable. It soon falls to a safe point, often without treatment. The seriousness of the febrile movement is to be judged by its duration and by the coincident nervous symptoms. Conditions leading to an unfavorable prognosis are irregularity in the appearance and progress of the eruption, especially if associated with severe febrile, pharyngeal, nervous, typhoid, collapsic, or gastro-enteric symptoms. Nephritis, especially if developed early, and the consequent dropsy, if the latter invade the serous cavities, are most unfavorable.

**Prophylaxis and Disinfection.**—The first thing in every case is the thorough isolation of the patient. Every child in the house not made immune to the disease by reason of a previous attack, should be sent away. Those remaining should be forbidden to attend school. The patient should be put in a room on the top floor of the house, where he will be farthest removed from those not in actual attendance upon him. Every article not necessary for the sick one's comfort should be removed. Carpet and drapery should not be permitted. All books and papers, toys, etc., used for the amusement of the patient, should be destroyed immediately after recovery. All bedding and clothing which are incapable of disinfection by boiling or exposure to dry or steam heat, should be burned. The thought of again using a mattress which has once served a scarlet fever patient should never enter the mind.

Many directions respecting the disinfection of the patient after the completion of desquamation have been given. In view of the ignorance which as yet prevails respecting the characteristics of the scarlatinous germs, these must be looked upon as unreliable. There can be no doubt, however, that the cast-off epidermis floating in the atmosphere forms an element of danger to others. It is wise, therefore, to anoint the body with some mild oleaginous substance, as cocoa butter, and wash care-



fully. Before throwing the washings away, they should be rendered innocuous by dissolving therein some powerful germicide, probably corrosive sublimate in the proportion of 1:1000. The scalp should be thoroughly shampooed, for it is well known that the scales are very liable to accumulate in that locality. In the case of females with long hair thorough disinfection without previous removal of the growth is difficult.

Articles not burned should be subjected to steam heat for a prolonged period. This, of course, can only be done where a large disinfecting apparatus is available.

When the patient has entirely recovered, the paper should be removed from the walls. The latter should then be thoroughly scrubbed with a solution of mercuric chloride, and whitewashed or calcimined. The flooring should be rubbed down and scrubbed and the woodwork thoroughly washed and repainted. Prior to all this, the windows should be thrown wide open and the room aired. It has been recommended that sulphur be burned in the room. Numerous experiments with this agent prove apparently the inutility of this method of disinfection so far as scarlatina is concerned.

The diet should be liquid, consisting mainly of milk, though soups may be given. The clothing should be of the lightest possible character consistent with proper protection. The greatest care should be exercised lest the child take cold. The room must, therefore, be kept comfortably warm. Water may be given *ad libitum*. Great cleanliness of the patient should be the rule. This may be secured by daily tepid baths.

*Belladonna* has been recommended as a prophylactic by both schools of medicine. It is probably, however, without value. Faith in its powers should not lead to carelessness in enforcing the prophylactic measures above recommended.

**Treatment.**—Prior to and during the early stage of the eruption, viz., before local phenomena attract special attention and treatment, there are a few remedies from which we are accustomed to select, and they are *aconite*, *belladonna*, *gelsemium*, and *veratrum viride*. *Aconite* for sthenic cases with restlessness and brain excitability. *Belladonna* if drowsy, dull, sleepy, and less arterial tension than in the *aconite* case. *Gelsemium* if the mental state is rather dull than active, and the patient is already prostrated, disinclined to exertion, and pulse feeble. *Veratrum viride*, if the case is not improved under *aconite*. Of either of the first three of these medicines, administer, of the first decimal dilution, ten drops in four ounces of water, teaspoonful doses hourly. Of the *veratrum*, one drop of the tincture each hour.

If thirty-six hours pass and the eruption does not develop, place the patient in a water bath at 100° F. for ten minutes. Repeat the pro-

cedure in three or four hours, if there is still a non-appearance of the eruption. If this second bath does not secure the desired end, and especially if the temperature is high (above 104.5° F.), the patient becoming more drowsy, energetic means must be at once adopted. Up to this time, the patient has been simply sponged, now the cool bath must be adopted at a temperature of 80° F., cooling the water gradually down to 65° F. if required. This low temperature is indicated if the fever does not fall sufficiently. This is determined by the thermometer placed in the rectum. If the cool bath fails, or if it reduces the temperature the symptoms of cerebral depression remaining, cold water must be used in a manner to secure its stimulating influence upon the nerve-centres, as well as its antipyretic effect. For this purpose water at from 60° to 50° F. may be dashed upon the face and upper part of the trunk, while the patient is in the bath. I have found excellent results from placing the patient upon a rubber blanket suspended properly or held by several persons, and dashing, spraying, or pouring water upon him. At certain seasons of the year, when the temperature of the water is about right, a hose may be attached to the water spigot, and the treatment carried out with little trouble. It is a good plan for the physician to have made a hose attachable to any washstand, so as to permit the mixture of the hot and cold water in any proportion, and thus securing that degree of temperature that may be desired. The importance of this method of treatment, not only as a means of reducing dangerous elevations of temperature, but as a stimulant to the depressed nerve-centres cannot be overestimated. The latter influence is decidedly the more important. It requires good judgment in order to properly carry out this plan, as well as to select proper cases for its application. It is better not to begin it unless the temperature is about 105° F. or higher, and not unless it persists at this high point for twenty-four hours, and is accompanied by increasing cerebral symptoms. After the supervention of this condition, it is generally useless to persist in the administration of any of the quartette of remedies recommended for the early fever.

It is at this period that we prescribe *rhus toxicodendron*, a remedy too frequently overlooked, although it has been most strongly recommended. No medicine with which I am acquainted is as generally valuable in scarlet fever. The high temperature, drowsiness, ulcerated throat, swollen glands and cellular tissue at the angle of the jaw, the red glazed tongue, and any form of eruption, if the remedy is suitable for the general condition, restlessness, perhaps headache, and general pain; these, and many other well-known indications, show its applicability to cases of this disease. Excellent results follow the use of extremely small doses. I prescribe *rhus tox.* 2x dilution, ten drops in four ounces of water, giving it in doses of one teaspoonful at intervals of from one to three hours as required.

During the past few years I have in many cases employed the *iodo-hydrargyrate of potassium* from the beginning of the attack and with most satisfactory results. Drop doses of a ten grain alcoholic solution every hour. Of all medicines administered it has influenced the course of the disease most positively. Its effect upon sore throat and glandular symptoms is decided. It is possible that its value is largely due to its action as a germicide.

If the nervous symptoms assume a high grade, if the intensity of the poison is such as to lead to threatened paralysis of the cerebral functions, that is, a tendency to coma or convulsions, and further, if these symptoms develop in excess of all others, *rhus* will prove of little use. *Ailanthus*, *camphor*, *hydrocyanic acid*, *cuprum* and *zinc* have been recommended for this condition and are all remedies of power. *Farrington* reports a severe case cured by *ailanthus*. The child lay in a stupor with mouth wide open, swelling of the throat both inside and out, acrid discharges from the nose and mouth, and a dark red or purplish rash mixed with petechiæ. This remedy differs from *arum* mainly in the association of the symptoms with stupor.

*Camphor* is to be utilized when there is profound prostration and the body is bathed in a cold sweat. Under *hydrocyanic acid*, the rash is livid from the very beginning, is interspersed with petechiæ, and the throat is œdematous. Under *cuprum* and *zinc* the mental symptoms and convulsive phenomena are prominent features. *Apis mellifica* has quite a sphere of utility in scarlet fever. In the beginning it is indicated when the fever runs high, associated with great nervousness. Throat symptoms are prominent. The patient suffers from a burning stinging raw feeling in the throat, which is much swollen and œdematous. Prostration is great and comes early in the course of the disease. The rash is bright in color, but is apt to be of the miliary type. Symptoms of meningeal irritation or actual inflammation also indicate its use.

In severe cases of the anginose variety, *belladonna*, *apis*, *mercurius*, *mercurius cyan.*, *mercurius biniodid*, *cinnabaris*, *lachesis*, *arsenicum*, and last, but by no means least, *rhus*, may be called for. The symptoms of *lachesis* are strongly suggestive of this condition. The rash is imperfectly developed, and of a dark purplish hue. The cellular tissue of the throat is inflamed and threatens suppuration. The cervical glands are swollen. The tonsils are covered with a dirty white membranous deposit. Hæmorrhagic tendency may exist as revealed by epistaxis. There is marked sensitiveness of the surface. This hyperæsthesia is not the result of inflammation.

Suppurative inflammations and ulcerations call for *hepar*, *silica*, *sulphur*, *mercurius*, and *calcareæ*, according to circumstances. When gangrenous changes threaten, *arsenicum* is probably the most promising remedy.



The otitis calls for most careful supervision. If cerebral symptoms threaten, and it is better to take action before this, the membrana tympani should be incised and the pus pent up within the middle ear evacuated. If drainage is good, and the constitution of the patient is not depraved, this will prove all sufficient. In some cases the otorrhœa persists. The ear should then be cleansed by careful wiping with absorbent cotton, the physician himself attending to this, and peroxide of hydrogen instilled. The packing with boracic acid in impalpable powder should be reserved for cases resisting this treatment, care being taken that the thickness of the discharge and the clogging of the meatus do not interfere with free drainage. During the existence of active inflammation cleansing and instillation of peroxide of hydrogen should constitute the whole treatment. Of remedies, *rhus*, *belladonna*, *hepar* and *mercurius* are suitable.

The renal complication calls for the exercise of the greatest care in the management of the patient's diet. Milk should be, if possible, the only thing administered. The patient should be kept at absolute rest in bed. Pure water should be drunk freely. The uræmic and other special symptoms should be treated on the same principles as recommended under the head of acute nephritis. Above all things the patient should be carefully kept from chill. The room must be maintained at a temperature of 70° F. or more. Unless contraindicated, *aconite* should be administered, dissolving five drops of the tincture in four ounces of water, and giving teaspoonful doses hourly. If prescribed sufficiently early, it may cut short the whole trouble.

For the fully developed nephritis *cantharis* enjoys the highest reputation, and has been praised even by old school authorities, probably on Ringer's and Lancereaux's recommendations. It is especially adapted to cases in which desquamation has been free, and uræmia threatens. It is not necessary that the bladder symptoms of the drug be present. The best results from *cantharis* are obtained from the administration of quite material doses, that is by mixing ten drops of the tincture in four ounces of water, and giving one teaspoonful of the solution every one to three hours as required.

*Arsenicum* is Hughes' favorite remedy for post-scarlatinal nephritis. It is adapted to less acute forms than is the preceding drug.

## MEASLES.

**Synonyms.**—Rubeola; morbilli.

**Definition.**—Measles is a specific contagious disease, probably dependent for its existence upon a special micro-organism as yet undiscovered, and characterized by catarrhal inflammation and a peculiar eruption, the former involving especially the mucous membranes of the respiratory tract.

**History.**—Measles has undoubtedly been recognized and described for many centuries. Ahrun, a Christian priest and physician, who lived in Alexandria in the seventh century, mentioned it. It was also described by Rhazes and Avicenna under the name of hhasbad. It was given the name of "morbilli," which means little plague, by the Italians. This latter name clung to it until the latter part of the eighteenth century, when Sauvages first called it "rubeola." This latter term was at once adopted by Cullen and Willan, and came into general use in England. Of late, however, it has been largely discarded. It certainly is a name that leads to confusion of ideas, notwithstanding the etymological reasons for its adoption. Rötheln, an entirely different affection pathologically, is occasionally referred to as "rubeola."

Measles seems to have been introduced into America much earlier than any of the other exanthemata, its appearance here being contemporaneous with the advent of the earliest settlers.

Measles is almost invariably regarded by the profession and laity alike as a very mild disease, one exciting but little anxiety or concern. This feeling of ease respecting it is only warrantable when proper hygienic conditions prevail and are made operative, and the subjects of the disease possess good general health. Epidemic after epidemic has been recorded in which measles has proved itself capable of being anything but a benign disease. Occurring epidemically among armies and in crowded communities, it may become a veritable scourge, scarcely inferior in gravity to many more dreaded disorders. In many camps in which it has made visitations, nearly one-half of those attacked have succumbed. It is said that during the Brazilio-Paraguayan war, measles destroyed nearly one-fifth of one of the armies in less than three months, a result due, however, not to the severity of the epidemic, but to the lack of proper food and shelter.

**Etiology.**—Measles, like all other forms of acute infectious fevers, is undoubtedly caused by a specific virus, the nature of which has not yet been determined. The specific element exists in all the mucous secre-

tions, the blood and the epidermis. Inoculations performed with blood taken from measles cases have succeeded in transmitting the disease in almost every instance. The period during which the discharges, etc., retain their virulence seems to be very short as a rule, for inoculations performed with blood ten days old have frequently failed. Analogy leads us to ascribe the origin of the disease to the action of a germ, no one at the present day making any claim for the *de novo* origin of measles. Numerous micro-organisms have been discovered in the secretions of measles patients, but the evidence that any one of these is really the cause of the disease, is yet wanting. The most recent of these discoveries of the so-called measles bacillus is that of Canon and Pielicke, who, after the investigation of a large number of cases, described what was apparently the ultimate etiological agent in the production of measles. Josias and Laveran have since then made independent investigations covering the same field, and have failed utterly to substantiate the results of Canon and Pielicke.

The majority of severe epidemics of measles occur during the cooler months of the year. Still the disease is not an infrequent one at other times. The influence of season is often imprinted on the character of the complications. Thus, during the cold and changeable weather of winter and spring, the catarrhal symptoms are severer, if not absolutely dangerous; while during the hot months, diarrhoea and intestinal complications prevail.

When once an epidemic of measles is started, its spread takes place with great rapidity. Very few of a susceptible age who have not been protected by a previous attack escape. These epidemics appear at rather regular though long intervals, the period of comparative immunity being due in all probability to exhaustion of susceptibility by reason of the populace having obtained tolerably complete protection by previous attacks of measles. The contagiousness of the disease is such that the time elapsing between the inception of an epidemic and that when it is at its height is very short in comparison with that observed in the case of epidemics of other eruptive disorders. The very rapid spread of measles suggests that the poison giving rise to it is of a very portable nature, perhaps very readily diffused through the atmosphere. The probability of this atmospheric spread is made a certainty by the occurrence of measles in subjects strictly quarantined, and who have not come in contact with patients suffering from that disorder. It is believed that the poison gained admission to the body by the respiratory tract, exciting there a catarrhal inflammation as a primary manifestation. Whether the catarrhal inflammation of the alimentary tract which sometimes exists is due directly to the taking of the poison into the stomach, or is caused less directly by constitutional influences, is as yet a mooted question. Active though the specific cause of measles be, it is neverthe-



less readily destroyed by free exposure to air, as shown by the ease with which fomites lose their dangerous property by airing.

Measles is contagious in all its stages. Just as soon (some say even sooner, while the incubative stage is still on) as the catarrhal symptoms appear, the patient becomes a source of danger to others. The intensity of the contagion is such that one can scarcely hope to prevent the spread of the disease when it has once gained an entrance into a household or an institution.

No age is exempt from measles. It is, however, a matter of general observation that comparatively few cases occur after the age of puberty. It is doubtful, however, if this immunity is the result of age; the claim being that adolescents and adults escape infection because of attacks of measles during infancy or childhood. Nursing infants of less than six months of age are rarely affected. Time after time has it been observed that these tender sucklings escape, while every other child in the house becomes affected.

Measles may attack the foetus in utero. In the cases recorded in literature the mother had suffered from the disease but a few days before delivery. Those observing the cases incline to the view that measles occurring thus, produces an endometritis and consequent expulsion of the child.

The possibility of measles attacking the same individual more than once has been much discussed. The general opinion favors the view that such recurrences do occur, but that they are very rare. The possibility of error is great, owing to the marked similarities presenting themselves between measles and rubella or r  theln. Neither of these diseases protect from the other. All reports of second or third attacks should be received with considerable doubt unless all the seizures have taken place under the observation of a single competent authority. The view taken by Osler that recurrences of measles are more frequent than of the other eruptive disorders is supported by but few authorities.

A specific contagious property is believed by some to be possessed by the ophthalmia and the broncho-pneumonia of measles, because when either of these complications has occurred in a given epidemic, numerous other examples of that complication appear in the neighborhood.

**Pathology and Morbid Anatomy.**—As in the other infectious diseases, the blood, in measles, is altered in the direction of lessened coagulability; it is dark and fluid. Among the special tissue changes, those found in the surface structures, the skin and mucous membranes, are the most prominent. On the skin, we find a papular dark red eruption, while the mucous membranes are rarely found free from the evidences of a more or less intense catarrh. The catarrhal inflammation begins in the upper respiratory tract, but quickly extends to the bronchial tubes. The intensity of the inflammatory changes vary greatly in different

cases. In a few instances, it may be strictly localized in one portion of the respiratory tract, *e.g.*, in the larynx. The affected membrane is hyperæmic, covered with increased secretion, and in some cases is quite cedematous. This last-mentioned condition occasionally constitutes a very dangerous feature, particularly if occurring in the larynx. The various forms of bronchitis and of pneumonia (especially the catarrhal variety of the latter) are quite frequently present. Collapse of the lungs sometimes occurs in young children. In cases meeting death before local lesions or complications have developed, it is common to find the organs generally hyperæmic and softened. The intestinal tract sometimes exhibits marked lesions, a general catarrhal process, or extensive follicular changes in the intestines, even ulceration is not uncommon. Miliary tuberculosis, general, or of the lungs, pulmonary collapse, pulmonary œdema, effusion into the pleural or other serous sacs, pericarditis, diphtheritic inflammation in the pharynx, and inflammatory changes in the kidneys, are some of the less frequent associated conditions.

**Clinical Course.**—As in the case of the eruptive diseases already studied, the course of measles may be best considered by dividing the disease into the stages of incubation, invasion, eruption and desquamation. The *stage of incubation* is generally from twelve to fourteen days duration. This has been positively determined by inoculation as well as through a study of its introduction into countries where the disease has not previously existed. It may vary, however, within the limits of one and three weeks. Cases in which this stage has lasted longer than three weeks have been reported; but it must be said of them that they lack authenticity. The stage of incubation is free from symptoms—although those old enough to take note of their conditions complain of loss of appetite, lassitude, etc. The *stage of invasion* is ushered in by febrile phenomena,—perhaps by a chill and the ordinary accompaniments of febrile attacks, *viz.*, nausea, vomiting, headache and pain in the back and extremities. With these manifestations of fever, the symptoms indicative of catarrhal inflammation of the upper respiratory tract, *viz.*, sneezing, coryza, soreness and rawness of the throat, redness of the eyes, and lachrymation appear. The catarrhal inflammation increases, and with extension to the larynx and bronchial tubes a more or less troublesome cough appears. This is often of a hoarse or croupy character, and is soon after its appearance attended with expectoration, but which is usually small in quantity. Diarrhœa is occasionally present. By the fourth day of the disease, the eruption appears, first upon the face, about the ears, upon the forehead or cheeks, and gradually extends to the trunk and extremities. By the sixth day the rash and the general symptoms of the typical disease attain their highest degree of development. During the stage of eruption the fever and catarrhal symptoms continue, and diarrhœa, if not already present, is frequently added to the symptomatology. About the sixth or the

seventh day the symptoms begin to subside rapidly; the fever speedily diminishes, the eruption fades, and the catarrhal symptoms disappear. The marked diminution of all symptoms at this time has led to this being called the stage of decline. The most marked feature of this stage is a slight degree of desquamation.

**Analysis of the Symptoms.**—The symptoms of measles as above outlined require further critical consideration. The prominent ones of the stage of invasion have been stated to be the fever and the catarrhal symptoms. To these we will first direct our attention.



FIG. 10.—TEMPERATURE CHART OF A CASE OF MEASLES.

**THE FEVER.** The accession of fever in measles is generally sudden, and always rapid. The initial rise is rarely below 102° F., and frequently as high as 104° F. Mild cases may arise, however, in which it does not go much over 100° F., thereby escaping detection in the presence of poor observers. It may be preceded by an actual chill or mere sensations of chilliness. Convulsions occasionally mark its advent, but are more common to the period just preceding the eruption. Attempts have been made without result, however, to assign to these convulsive phenomena of measles, characteristics differentiating them from similar conditions associated with other diseases.

The fever reaching its height rapidly begins to recede on the second



day, indeed, it may fall to normal or a degree or so above that point. From this time, until the appearance of the eruption, it remains a subordinate feature of the illness. If it has any special feature, it is that of irregularity. With the fourth day of the disease and the appearance of the eruption, the fever again rises rapidly and still higher than at the beginning of the stage of invasion. Remaining at its acme for from twenty-four to thirty-six hours, it disappears by crisis. Any prolongation of the fever beyond this time, is a tolerably certain indication of some complication.

The fever in the initial stage of the disease may sometimes be associated with a soporous condition, thus leading to uncertainty in diagnosis. This was well illustrated in a case admitted to the Hahnemann Hospital one year ago. The patient had received a penetrating wound of the orbit. Three days after her admission, there was sudden fever with stupor, associated with cough. All indications of focal brain lesions were absent. The nature of the case was therefore regarded as uncertain. Consciousness was regained with the appearance of the characteristic eruption of measles.

**CATARRHAL SYMPTOMS.** The catarrhal inflammation is usually discovered first in the nose, manifesting itself there by a sensation of dryness, which is rapidly followed by sneezing and coryza. Pain in the frontal sinuses and rawness in the pharynx are associated. The eyes become inflamed and injected, and free lachrymation frequently sets in. The catarrhal process rapidly extends to the larynx, trachea and bronchi; then cough becomes a prominent and troublesome symptom. An examination of the throat on the second day reveals the presence of a number of dark red spots about the uvula and the soft palate. These may become confluent and give the parts a diffuse erythematous discoloration. This condition, in conjunction with the symptoms already enumerated, is regarded as highly characteristic of measles. Indeed, it, with the catarrhal symptoms, assumes especial diagnostic importance in the case of measles occurring in the colored race.

With the appearance of the eruption the catarrhal symptoms increase in intensity. In a fair proportion of cases, the cough becomes the really troublesome symptom, that is to say, the one requiring attention. The bronchial involvement does not usually extend lower than the larger tubes.

**THE ERUPTION.** This almost invariably appears on the fourth day of the disease. Exceptionally, it may be observed as early as the third or as late as the fifth. It makes itself known first upon the face by the appearance of papules, rose-red in color and but very slightly raised above the general cutaneous surface. These spots range in size from one to eight-twentieths of an inch in diameter and are grouped together in clusters. They are irregular in outline, and their margins are sharply defined from the surrounding healthy skin. Sometimes they assume a

bluish tinge. They may remain discrete, or they may become confluent; especially does the latter occur to the eruption on the face. From the face the rash of measles spreads to the trunk and extremities, but it is never as profuse on the part first named. The rash can be made to disappear momentarily by pressure with the finger.

Sometimes, small extravasations of blood may be observed in the midst of the characteristic eruption. This condition is doubtless due to a temporary alteration in the structure of the minute bloodvessels permitting of an escape of their contents.

The rash disappears first upon those parts on which it first appeared; with its fading there is a branny or furfuraceous desquamation developed. This latter symptom is usually in direct proportion to the intensity of the exanthem, though not necessarily so. It is most marked upon the face. It is apparently lessened by profuse perspiration, and frequent cleansing.

**Complications, Sequelæ, etc.**—These (complications and sequelæ) are much more liable to appear in cases showing previous ill health. The “strumous” diathesis especially, favors pneumonia and catarrhal developments. While the vast majority of cases of measles pursue the characteristic course already outlined, no disease is more notorious for the diversity of its complications and sequelæ, rare though these be. Cases running their course without the catarrhal symptoms have been observed (*morbilli sine catarrho*); other instances in which the rash is absent occur (*morbilli sine exanthemate*). Sometimes, fortunately indeed, very seldom, the patient is overwhelmed by the intensity of the poison (*morbilli siderans*). Another severe type of the disease is the so-called “black measles” (*morbilli nigra*), which is characterized by extensive hæmorrhages from the mucous membranes and into the eruptive points. This is a very dangerous form, and occurs almost exclusively in the cachectic and those living under unhygienic conditions. Anomalous conditions of the eruption are occasionally noted. The most frequent of these is its first appearance on some other portion of the body than the face. Sometimes the normal rash is interspersed with vesicles or bullæ. The latter are indicative of a rather serious condition. The catarrhal inflammations of the respiratory tract sometimes assume such severity as to constitute actual complications. Bronchitis is not at all uncommon, and as long as it involves only the larger tubes is not a serious matter. Extending to the finer bronchioles, it is a frequent cause of death, especially in the rachitic child or enfeebled infant.

Catarrhal pneumonia is the most common complication of measles. It presents no especial points of difference from the typical disease occurring under other circumstances. It is the most frequent cause of the mortality of measles. It is always to be suspected when, after the subsidence of the eruption, the fever persists. The origin of the pneu-

monia in measles is a matter for discussion. The prevalent view attributes it to exposure; others claim that it is the direct result of the specific poison. Sometimes the disappearance of the eruption is delayed by this complication.

The catarrhal inflammations of the eyes and tympanum at times assume quite serious proportions. Affecting the former organs, very troublesome conjunctival and corneal diseases ensue. Especially is this result liable to occur in dyscrasic subjects. Indeed, it is not uncommon for the various forms of serofulous ophthalmia to follow measles, and persist for a greater or less length of time according to the depth to which the constitution has been undermined. Ear troubles are as common as those of the eyes. Unlike the aural sequelæ of scarlatina, they are generally catarrhal and not suppurative in character. During their first stages, they are attended by the usual symptoms of catarrhal otitis media. They may result in permanent impairment of hearing. It has been said that from 8 to 10 per cent. of all cases of catarrhal deafness find their origin in measles.

Holt reports a very severe and fatal ulceration of the larynx occurring after measles in a child one year of age. At the autopsy the ulceration was found to have completely destroyed the vocal cords, and extended upward to the ventricles of the larynx, and downward for a distance of a quarter of an inch.

Digestive complications come next in frequency to those of the respiratory tract. Of these stomatitis and diarrhœa are especially prominent. The latter condition is especially apt to occur with considerable regularity in certain epidemics. It is very liable to set in as a result of laxative medication to relieve the constipation of the active stages of the measles itself. A very rare but exceedingly dangerous complication is noma. It occurs only in cachectic subjects.

Tubercular diseases of various organs have been observed to follow measles with such frequency as to suggest a definite relationship between the two. This relationship, however, must not be looked upon as one of direct cause and effect. The disease produces changes in the body offering favorable culture media for the tubercle bacillus, and thus rendering actively dangerous what would have otherwise been an innocent microbic invasion. For the same reason, tuberculous bronchial glands which had hitherto given no symptomatic evidence of their presence, become actively infecting after attacks of measles. The mistake of attributing all to tubercular infection must not be made.

Many of the severe forms of nervous disease following measles are the result of various processes; for not infrequently one meets with hemiplegias and epilepsies depending upon vascular disturbances, such as sinus thrombosis, or degeneration and rupture of walls of bloodvessels. Cases of myelitis and polyneuritis have been reported after measles.



Ozaena is quite a common sequela. Care must be taken, however, in view of the frequency with which children "poke" foreign bodies into the nose, that the so-called ozaena is really such, and not dependent upon local irritation.

Endocarditis is one of the less frequently recognized complications of measles. There is a growing conviction that this trouble occurs oftener than we have hitherto been willing to admit. It is not at all unlikely that many of the cases of unexplainable valvular heart disease in children have found their origin in attacks of measles. In the cases reported by Hutchinson, a rheumatic tendency existed in three.

Measles occasionally exerts a therapeutic effect upon certain pre-existent morbid processes. Quite a variety of skin diseases have been cured by its visitations, and it occasionally exerts a wonderful influence over certain nervous diseases, notably chorea and epilepsy. A most remarkable instance of this kind occurred in the Hahnemann Hospital of this city, under the care of Dr. Bartlett. The child had been subject to many convulsive seizures daily (often as high as sixty), which were greatly modified by a strict milk diet without any medication. The child contracted measles from a patient to whom reference has already been made. During the course of the disease, the convulsions increased in frequency. Since recovery, now six months, it has not had one seizure.

**Diagnosis.**—One of the most reliable data on which an early diagnosis may be based, is the knowledge of the existence of other cases of measles in the neighborhood, or of exposure to infection on the part of the patient. A correct opinion is not a difficult matter when the fully developed history of the cases is before us, for no condition simulates the sudden initial fever and its accompanying catarrhal symptoms, the drop in the temperature on the second day, and the reaccession of fever with the appearance of the eruption on the fourth. Notwithstanding this strong individuality of measles, it has been confounded with quite a variety of diseases, notably r  theln, variola, scarlatina and typhus fever. The differentiation from r  theln will be considered when treating of that disease. That from variola will be described later (see page 286).

*Scarlatina* is not characterized by the initial catarrhal symptoms, but by a well-marked and characteristic angina. The fever does not remit on the second day, and the scarlet fever exanthem appears much earlier than that of measles.

*Typhus fever* is characterized by severe and early prostration; a rash appearing for the most part on the trunk; profound impairment of the mental faculties; and the course of the accompanying fever. In every case, the fact that the patient has or has not had measles before is of value in reaching a conclusion. The dictum of Osler, that measles more than any other of the exanthemata is liable to recurrence, is not in consonance with the experience of most observers, and in view of the great liability to error, must be accepted *cum grano salis*.

**Prognosis.**—While measles is undoubtedly in general a mild disorder, the prevailing tendency of the profession is to regard it with increased respect, as fraught with great possible dangers. In private practice, among intelligent patients who follow physicians' instructions most rigidly, deaths are very rare indeed. No matter how severe the symptoms, providing no complications are present, recovery may be most confidently looked for. In asylums, "homes," and tenement-house districts, a very different picture is presented, measles here often being a fatal scourge. Statistics have been offered showing that the mortality of the disease among children under two years of age is 50 per cent., and over that age, 15 per cent. While these figures are undoubtedly too high, they show that measles must be regarded in the light of a serious disorder. Mortality statistics show that in many years more deaths occur from measles than from scarlatina, and yet the latter is a dreaded disorder. Adopting figures from the census of 1874, the death-rate from measles was 1.7 per 10,000 inhabitants in the United States. In certain parts of France it has reached 40 per 10,000.

As to the complications of measles, these are always serious, and especially does this remark apply to the broncho-pneumonia. A very large proportion of the victims of this complication die. Proper care and hygiene should prevent its appearance.

**Treatment.**—The first thing to be done when measles has invaded a household or an institution, is to prevent its spread. With the majority of people this is regarded as an entirely unnecessary precaution, owing to the ordinarily mild course pursued by measles, and the feeling that children must sooner or later be affected with it. The patient should at once be isolated in a well-ventilated room. Too little attention is paid to this detail of the sick-room, because of the fear of giving the patient cold from drafts; these should, of course, be avoided by the proper position of the bed or the placing of suitable screens about the patient. Fresh air not only serves to prevent spread of the disease by dilution of the poison, but it has a beneficial influence upon the respiratory tract. The bedclothing should be frequently changed and the body of the patient sponged daily.

The *diet* should be light but wholesome. With but few exceptions it should consist of milk only. If the patient is thirsty, he may be permitted to drink freely of water.

Careful *bathing* aids the functions of the skin, and is therefore a desirable measure. The use of soap and warm water every second day is advisable.

Owing to the ocular complications it has been customary to keep patients in a dark room. This measure is unwise, as the darkening of the room interferes with ventilation, a far more important measure. Better indeed to protect the eyes by the use of screens placed between the

bed and the source of light, or in extreme cases to cover the eyes with smoked glasses. It has been recommended in case of severe photophobia that cocaine or morphia solution be instilled into the eyes, but such a procedure is bad practice, as it dulls the sensibility of the cornea and permits the patient to expose his eyes to irritation, a thing that he would not do otherwise; and still more important, cocaine secondarily causes a paresis of the vessels and favors inflammatory action. Bacteriological examinations of the secretions of the mouth have shown that the pneumococcus is often present in large numbers. This indicates the necessity for careful attention to cleanliness of that cavity. The nurse should regularly, twice daily, wash the patient's mouth with water to which may be added some simple antiseptic, such as boric acid or listerine.

*Aconite*, in the opinion of most observers, is the remedy *par excellence* for the initial stage of measles pursuing a normal course. The fever, the photophobia and the coryza all suggest it. Hughes recommends that this remedy be given throughout the entire course of measles, and that if symptoms or complications call for any other remedy that it should be given in alternation with aconite. My personal experience has led me to consider *gelsemium* as a more effective remedy, giving it from the beginning in drop doses of the tincture, repeated every two hours. Of course, symptoms will frequently permit a clear differentiation between these remedies. For the early stage, *belladonna* is also a valuable medicine, being suggested by the character of the cerebral symptoms, the flushed face, the full pulse with little tension, and moist skin. Some prefer *veratrum viride* for this stage. In sthenic cases with pain, restlessness, sleeplessness and high fever, *phenacetin* in the first decimal trituration repeated hourly, may relieve promptly.

Quite a variety of medicines are employed for the catarrhal symptoms. Of these *pulsatilla* is the most prominent. It is best suited to cases with little fever. It affects favorably catarrhal inflammation of the ears and eyes as well as of the respiratory tract. In my experience it has not proven capable of coping with intense catarrhal inflammations, but rather with mild blennorrhœas. If the inflammation of the eyes and nasal passages is quite severe and secretion acrid, we may follow Pope's advice and give *euphrasia*. This is a valuable remedy and one, I think, little used in America. A most valuable remedy for the catarrhal symptoms of measles is the *bichromate of potash*. It is generally as efficient in the severe forms as *pulsatilla* is in the milder ones. It seems equally efficient in the ocular, aural and respiratory forms, the best effects being secured by giving two grains of the second decimal trituration every one to three hours. If the bronchial tubes endure the brunt of the attack and there is a heavy catarrhal expectoration, the *iodide of antimony* is suggested, and under the same circumstances if there is much dyspnoea, loud bubbling râles or numerous subcrepitant râles, *tartar emetic* or the



*arsenite of antimony* is to be preferred. When there is little secretion and a hoarse ringing cough, I can corroborate the statements of the late Walter Williamson as to the value of *drosera*; if ineffective in dilution, give of the tincture two to five drops every one to three hours as required. *Sticta pulmonalis* is an effective remedy when there is little or no secretion and an annoying hyperæsthesia. *Lachesis* may be considered if *sticta* fails. Opiates are much given for this form of cough.

Cases presenting symptoms of malignancy must have careful attention paid to their nourishment. Peptonized milk, koumiss, animal broths, eggs shaken with a little sugar and carbonized water, and the use of filtered and iced whey as a drink are suggested. Stimulants are generally demanded; wine whey, or brandy, or whiskey with whey, may be prescribed, and the same articles can be administered by the rectum, especially if refused by the stomach. Hot or cold packs may be useful for conditions of depression or high temperature. The urine should always be examined in these cases. For imperfect development or retrocession of the eruption, *camphor* has been most commended if collapsic symptoms are present; if the prominent symptoms are rather cerebral, *cuprum aceticum* or *hyoscyamus*, sometimes *stramonium*. If chest symptoms, *bryonia*, *tartar emetic*, *arsenite of antimony*. Hughes recommends *carbonate of ammonia*, first decimal, and in larger doses it is much recommended by old-school authors. Occasionally one meets with a well-developed typhoid state which is well combated by *rhus toxicodendron* or *baptisia*. For protracted catarrhal symptoms following upon measles *sulphur* is often indicated, and clinically very valuable (also the *iodides of arsenic*, *antimony* and *tin*). Otitis media occurring in association with measles is apt to be neglected or unskilfully treated by the general practitioner. As the treatment is the same as that detailed in the treatment of scarlatina it will not be reiterated in this place.

Gastric distress and vomiting may be treated with ice, a little iced champagne, and, perhaps, a mustard plaster to the epigastrium. The arsenite of copper in doses of one-hundredth of a grain (second decimal) is almost specific for this condition as well as for the diarrhœa. When laryngitis is present it should be treated as will be instructed for croup. Attempts to modify the fever by throttling it with antipyretics, if the disease is pursuing a normal course, are unwise, as high temperature *per se* is of but little consequence. Should the high temperature be due to a complication it is an important index and should not be interfered with. In these exceptional instances, however, in which there is hyperpyrexia and the patient seems to be suffering directly therefrom, it is good practice to resort to cold sponge bathing or other antipyretic measures as described at length in the article on typhoid fever. Should symptoms referable to the lungs persist, the great danger of development of tuberculosis must be borne in mind, and treatment suitable to incipient tuberculosis promptly

and persistently applied until all physical signs of bronchitis or pulmonary consolidation have disappeared.

The patient recovering from measles should not be allowed to leave his bed until all catarrhal symptoms are well controlled. According to the severity of the case this period varies from six to ten or twelve days. Before mingling with other persons the patient should be well bathed and dressed in fresh clothing. During the course of measles other children of the household must not be allowed to go to school. There is danger of conveyance of the disease germs to the houses of friends by the adult members of the family, a point, strangely enough, often overlooked.

## RUBELLA.

**Nomenclature.**—The prevalence of confused ideas relating to the nature and even to the actual existence of this disease, has given rise to an equally confusing nomenclature. It is known among the Germans as rubeola, roseola, and rötheln. The former of these terms is used by Thomas to denominate measles, and rötheln is a most inconvenient term for American and English use. French measles, German measles, bastard measles are terms in common use among the laity. Numerous other synonyms based on a hypothetical hybrid nature of the disease have also been advanced.

**Definition.**—Rubella is an acute infectious disease affecting mostly children, attended by early enlargement of the lymphatic glands of the neck and adjacent parts, fever, a characteristic eruption, and rendering the subject thereof immune from subsequent attacks of the same disorder, but not affording any protection whatever from either scarlatina or measles.

**History.**—Reliable evidence shows that rubella did not receive recognition as a distinct disease until 1740. Still it is claimed that the ancient Arabian physicians described it under the name of Hhamikah; and in the seventeenth century rossalia epidemics were described by the Italians. Up to within thirty years the disease has been denied a separate existence by many competent authorities, who looked upon it as either scarlatina or measles pursuing an abnormally mild course. Generally an exceedingly mild disease, it occasionally assumes serious proportions, so that at one period in Berlin it caused more deaths than did scarlatina and measles combined.

The identity of rubella as a distinct disease is now recognized on several grounds: (1) The contagion from rubella can produce this disease and no other. (2) Attacks of rubella protect from subsequent attacks of that disease, but offer no immunity whatever to scarlatina and measles. (3) It occurs in subjects who have had scarlatina or measles or both. (4) It has a characteristic clinical course of its own.

**Etiology.**—This disease may occur either sporadically or epidemically, the latter, however, by far the more frequently. It is eminently contagious, though less so than measles. It may be carried by fomites. Edwards refers to numerous cases occurring in immigrant children, contracted from sleeping on infected bunks in immigrant vessels. The contagion becomes more active by concentration, that is by the crowding of those sick with rubella within a given compass. It is believed that heat



and moisture favor the spread of the disorder. While thus stating a firm belief in the contagiousness of rubella, it is well to add that there are many who regard it as not at all or but feebly so.

Age is a powerful predisposing factor to infection with rubella. It is almost solely a disease of childhood, attacking boys and girls indiscriminately. Adults are not exempt; women are more frequently attacked than are men of the same age. Susceptibility to the disease appears to be lost after middle life is reached.

**Symptomatology.**—Rubella has a stage of incubation which has been variously estimated by nearly every author who has taken interest in the subject. It is especially noteworthy that individual writers do not state it as a definite period. It probably ranges between two and three weeks. During its continuance no symptoms are present, although it is believed by some that the disease is contagious at this period.

The stage of invasion is not marked by any characteristic symptoms. In nearly all but the severe cases, symptoms may not appear until just before the breaking out of the eruption. Fever may be present, but it is always slight. It practically never goes above 100° F., so that the exceptional cases in which it rises to 103° F. are generally regarded as curiosities. Other invasion symptoms are of the usual indefinite character—malaise, nausea, headache, etc.

Next comes the stage of eruption, and this may arrive without warning, the invasion symptoms escaping observation by their mildness. The characteristic symptoms of this stage are the glandular enlargements and the eruption. The former affect the cervical, suboccipital, submaxillary, and axillary glands especially. The rash first makes its appearance on the face and extends downward. It comes within a few hours of the initial symptoms. Indeed it may, as already stated, come without any premonition, the child being put to bed in the evening in the best of apparent health, and wakes in the morning in nearly full efflorescence. No part of the body is exempt from the eruption of rubella. It may be universal, or confined within very limited areas. Cases presenting the latter feature are mainly recognizable through knowledge of an existing epidemic. The color of the rash is usually a pale red or rose, lighter than that of measles. Still it may occasionally take on a deep-red or purplish hue. The rash itself has a punctated appearance, the central portion of each patch being darker in color than the remainder, and somewhat elevated above the surrounding skin. Individual patches present great irregularities in size and shape. The skin between them is hyperæmic. The duration of the rash has been variously stated by different authors. The majority give it but a short existence, twenty-four to forty-eight hours. Edwards states that in his series of cases the average was five days, the limits being two and fourteen days. He quotes numerous authorities to show the remarkable variations exhibited by the eruptive

period. He concludes that its average duration is three or four days. Desquamation may be absent or may take place by furfuraceous scales.

Mild catarrhal symptoms may be present or absent. When the former they never assume the intensity so common in measles. Slight coryza, conjunctival injection, bronchial cough and sore throat include them all. Œdema of the face has been observed in quite a number of cases.

**Complications.**—The complications of rubella are practically the same as those observed in connection with measles, those referred to the respiratory tract being the most frequent. Proper care should prevent their occurrence, for the tendency to depart from the typical uncomplicated type of disease is very weak indeed in rubella. Next to respiratory complications, we find in frequency, those of the gastro-intestinal tract, enteritis and muco-enteritis.

**Diagnosis.**—In the presence of an epidemic, the diagnosis of rubella is a very easy matter indeed. From measles we distinguish it by the mildness of the initial symptoms, the light rose-color of the rash, and the absence of marked catarrhal symptoms. A history of a preceding attack of measles is a valuable guide to a correct conclusion. Scarlatina presents far more violent symptoms, a perfectly smooth rash, and severe sore throat.

**Prognosis.**—The mortality of rubella is almost *nil*. As already stated, some epidemics of rather high mortality have been recorded. Why this should have been is not understood by present observers.

**Treatment.**—Notwithstanding the mildness of the disease, it should be shown sufficient respect to put the patient to bed, and surround him with all the hygienic measures that are accorded cases of measles and the other exanthemata. As to remedies, *aconite*, *pulsatilla*, *kali bichromicum*, *belladonna*, and *rhus*, furnish a list from which a good selection may be made.

## VARIOLA.

**Nomenclature.**—Commonly called smallpox; a name introduced late in the fifteenth century to distinguish variola from syphilis; the term varioloid being applied to a mild form of the disease, appearing in persons partially protected by vaccination.

**Definition.**—Variola is an acute infectious disease whose special characteristic is an eruption, appearing first in the forms of macules, and successively passing through papular, vesicular and pustular stages. The eruption runs a rapid course, and appears especially upon the uncovered portions of the skin, and upon the mucous membranes.

**History.**—The current belief of the existence of smallpox prior to the Christian era is strong, but there is no reliable evidence in support of it. The earliest accounts of its ravages are by writers in the sixth century. In 581 Gregory described a scourge which passed over Southern Europe, and which, from its description, must have been smallpox. He carefully differentiated this epidemic from the bubo-plague, which also invaded Europe a year later. Rhazes, of Bagdad, described with great clearness the important features of the disease in 900. England was invaded in 1241-1242. It appeared in Mexico in 1527; in South America in 1554, at about the time slaves were carried there from Africa. It did not reach the United States until 1649, when an epidemic started in Boston. All these epidemics were of remarkable severity, the victims being numbered by millions; the first Mexican epidemic, for example, is said to have carried off three and a half millions of people. Since the wonderful discovery of Jenner in the latter part of the last century (1798), however, the disease has yearly claimed fewer and fewer victims, until during the past twenty-five years, epidemics of any noteworthy severity are all but unknown. The most important within the last decade was that occurring in Montreal in 1885, and was solely due to the opposition of the French Canadians to vaccination.

**Etiology.**—Variola is highly contagious, few persons unprotected by vaccination escaping the disease if freely exposed. Occasionally, however, a person is met who neither contracts the disease nor is susceptible to the vaccine virus. This immunity from the contagion may be but temporary, *i. e.*, it may exist during one epidemic and not during another.

The contagium is given off by the skin and lungs of the sufferer. The fine epithelial scales which are thrown off the skin so freely are supposed to be the most important agents in conveying the specific ele-



ments of the disease. These small particles attach themselves to clothing, furniture, the walls of the room, and even to domestic animals. The stage of the disease at which contagion is possible is still a matter for discussion. It is generally admitted, however, that the disease may be transmitted at any time during its course, but is most dangerous to others when suppuration is about to commence. In rare instances persons have been known to contract the disease from patients in the incubative stage, before even a vestige of the eruption is present.

The poison retains its vitality for a long period of time, indicated by the fact that the clothing of persons who have suffered from the disease, and which has been stored for a considerable period of time, has given rise to fresh cases upon being unpacked in a new neighborhood, one which had hitherto been free from the disease. During the epidemic prevalence of smallpox, public conveyances of all kinds, mail matter, money, etc., are active agents in its spread. It must not be forgotten that varioloid is but a modified form of smallpox, and capable of infecting a well person with the most malignant variety. The nature of the specific cause is as yet undetermined. It is known, however, that it exists in its most active form in the contents of the vesicles, especially at that time when the contained serum is about to become purulent. It is doubtful, however, if the poison is contained in the natural secretions of smallpox patients, for inoculations performed with the saliva, urine, expectoration, etc., have failed to reproduce the disease. The saliva and expectoration must, however, remain always under suspicion, because the mucous surfaces are liable to the eruption, the contents of which may well contaminate the ordinary mucous secretions. It has been claimed that inoculation with the blood of a variolous patient will not spread the disease. Experiments on the lower animals and the infection of the fœtus by the mother show the falsity of this belief.

Smallpox attacks persons of all ages and of both sexes alike. The disease is especially frequent and fatal in children, probably because there is more susceptible material to attack, in the early years of life. As previously stated, the fœtus in utero may contract the disease from the mother; children have been born with the eruption in various stages of development or with well-marked cicatrices. The period of intra-uterine life at which variola may be contracted, is any time between the fourth month and birth. In all such cases the fœtus is infected with the disease by the mother. Some few cases apparently antagonizing this idea have been reported, for children have been born scarred with smallpox, and yet it was asserted that the mother did not suffer from that disease during her pregnancy. Under such circumstances it is highly probable that the maternal attack was so light and the eruption so indistinct as to escape recognition, the fœtus enduring the brunt of the disease. Curschmann reports such a case.

Smallpox has been highly fatal among certain people, especially among the aborigenes of a country. It has been the great scourge of the American Indian. The colored race shows a most remarkable susceptibility to the contagium, and to severe attacks of the disease. This is probably due to their unprotected condition from lack of vaccination and their usually bad hygienic and sanitary conditions, for these favor the development of any contagious disease.

Various observers (Weigert, Cohn, and Klebs) have described micro-organisms, especially in the pock, which these observers have supposed to bear an etiological relationship to the disease. They have thus far, however, failed to bring forth satisfactory evidences showing that such a relationship exists.

Much discussion has taken place over the possibility of existing diseases acting as a preventive of smallpox, many believing that immunity is thus temporarily created. Such a belief has but a limited foundation in fact. Certain acute infectious diseases, notably scarlet fever, measles and typhoid fever, seem to protect the sufferer from the variolous contagium during their continuance. Other acute diseases, and all chronic ailments offer no protection whatever.

The severity of the disease varies greatly in different years and in different epidemics, facts which are capable of a very simple explanation. We are not obliged to fall back on the ancient superstition that smallpox epidemics return at regular periods. There are always some cases present in large communities, such as those of our great cities, but careful health regulations, and the thorough practice of vaccination, keep the disease from spreading. Long freedom from an epidemic leads to public carelessness, with neglect of vaccination and consequent increased susceptibility of the population to the disease. An epidemic is the consequence. The people, thoroughly frightened, resort to efficient protective measures (vaccination), and the epidemic is soon at an end for the want of suitable material.

**Pathology and Morbid Anatomy.**—The vesicle of smallpox presents a reticulated structure. This accounts for the fact that it does not collapse when punctured, because only one of its little compartments is opened. Weigert has carefully studied the eruption, and states that a section of the papule, just as vesiculation is beginning, exhibits in the *rete mucosum* a small area of granular cells which are not affected by the staining fluid, these cells being in a state of coagulation necrosis resulting from the action of certain micrococci. About this area a zone of inflammation is established. The necrotic area represents the future point of umbilication. Scars ensue when this inflammatory process or rather its result—destruction of tissue—extends into the cutis vera and deeper structures. The pustules have been found upon all portions of the mucous membrane, but especially upon that covering the mouth,

pharynx, larynx, bronchi, œsophagus, etc. The lymph follicles in the intestinal mucous membrane are often swollen. In the larynx and bronchial tubes the eruption is sometimes attended by a deep-seated inflammation involving even the cartilages. A croupous exudate has been found in the larynx. The eruption in this region does not present the typical appearance, but rather irregular ulcerative erosions. The lungs are frequently hyperæmic. Lobular pneumonia is common; lobar pneumonia unusual. The spleen is much enlarged. The liver is not rarely the seat of fatty changes. A diffuse variety of hepatitis has been repeatedly seen. This is attended by intense congestion and rather diffuse suppuration.

The vascular system does not manifest uniform changes. No regular alterations in the composition of the blood are found. The heart may be fatty, or simply have undergone a parenchymatous change. The lining membrane of the vessels may present fatty areas. Active inflammation of the cardiac membranes is unusual. The urine is occasionally albuminous, this being at times due to nephritis—which is a complication very liable to ensue during convalescence. Orchitis is quite common.

In the hæmorrhagic variety of smallpox, hæmorrhages into or from any of the tissues of the body have been reported by different observers, and need not be stated in detail.

**Clinical Course.**—The incubation stage is of ten days to two weeks duration. In a few cases, the period may be shorter or longer. Prodromal symptoms are not common. Still, some few cases are observed in which this period is characterized by certain symptoms indicative of a general constitutional disturbance. As there are marked differences in this malady as developed in different individuals, various classifications of it have been suggested. The following is the one generally adopted:

- I. VARIOLA VERA. This being divided into (a) *discrete*, and (b) *confluent*.
- II. VARIOLA HÆMORRHAGICA. This being divided into the purpuric, the hæmorrhagic and pustular forms: (a) *purpura variolosa*, (b) *variola hæmorrhagica pustulosa*.
- III. VARIOLOID. Variola in a person who has been partially protected by vaccination.

The period of incubation has been accurately determined by means of inoculation of the disease practised before the introduction of vaccination. In such inoculation, six to nine days generally elapsed between the introduction of the virus and the appearance of symptoms. When contracted in the ordinary manner, fourteen days may be required for the development of symptoms. Longer and shorter periods of incubation are reported, however.



The stage of invasion is marked by one or more chills, with a rise of temperature from 102° F. to 103.5° F. during the first twenty-four hours. The chill of smallpox is very severe, more violent perhaps than the chill of any other eruptive disease. It may consist of one long or a succession of shorter chills. The temperature continues to rise until the evening of the third day, when the mercury may attain a height of 105° F. to 106° F. The pulse becomes full and increases in frequency to from 120 to 150. Attending these early fever symptoms are severe headache, backache, and pain in the extremities, nausea, vomiting, sore throat, photophobia, flushed face, beating carotids, and perhaps delirium. By the third day, often on the second, the tonsils and throat generally present redness and other evidences of inflammation. This inflammation may extend to the communicating mucous surfaces, especially to the larynx, when hoarseness and cough appear.

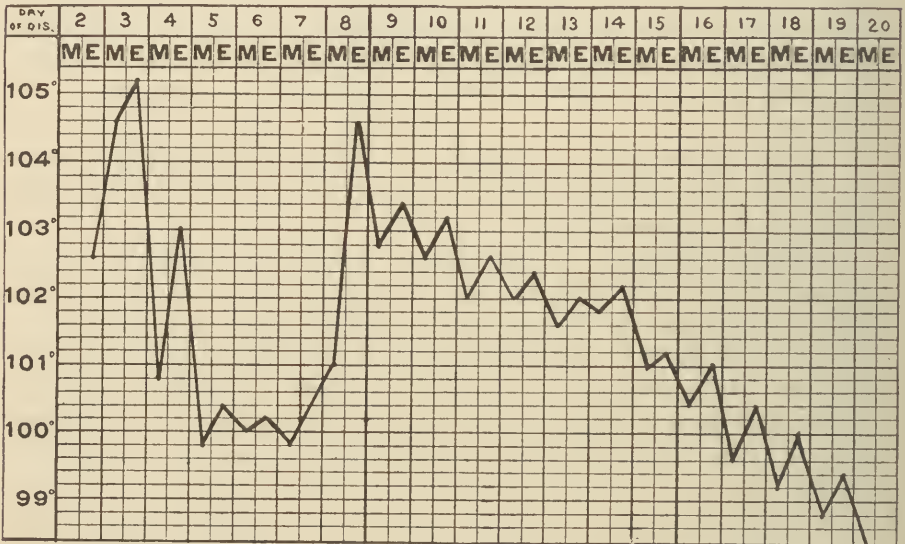


FIG. 11.—TEMPERATURE CHART OF A CASE OF SMALLPOX.

Although the above-named invasion symptoms apparently present nothing especially diagnostic, they are invaluable and full of significance in the presence of a smallpox epidemic. The *headache* and the *backache* are especially significant. Both of these symptoms are severe. The headache especially is remarkable for its constancy, and appearance within a few hours of the onset of the fever. The backache was regarded by Rhazes as an important diagnostic symptom. Its severity is usually proportionate to the intensity of the disease. It disappears on the advent of the eruption.

In some cases the gastric symptoms assume prominence, and vomiting becomes so severe as to constitute a source of great suffering.

Nervous symptoms are sometimes the special feature in the stage of invasion. Thus in children convulsions not infrequently mark the onset of the disease. In other cases, syncope and deep coma are observed. Many such cases are at once overcome and die from the intensity of the poisoning, before the eruption has had an opportunity of appearing.

During the stage of invasion there occasionally appears a rash which may be of either an erythematous or a petechial type. The former of these may assume one of two forms, the "scarlatinal" or the "measly." It does not show any special preference for any particular part, indeed it may spread over a large portion of the body. It is believed that many of the cases of so-called combined scarlatina and smallpox, and measles and smallpox, which were so frequently reported in former years, were but instances of this type of initial rash. The objective appearances of the erythematous rash present nothing characteristic of this disease. Occasionally it is interspersed with petechiæ. It is a symptom indicating a light attack of the disease. Indeed, it has been generally observed that the true variolous eruption exhibits but little tendency to affect the parts on which the erythema has appeared.

The initial petechial rash is not a sign of favorable import. It is especially apt to affect the lower portion of the abdomen and the inner surfaces of the thighs.

The frequency with which the initial rashes appear varies greatly in different epidemics. In some, they are remarkable for their constancy; in others, they are equally so for their rarity.

The severity of the invasion symptoms is not a positive guide to the future seriousness of the case, severe early symptoms being often followed by a very mild development of the disease, and *vice versa*. Mild invasion symptoms usually, however, indicate a mild course.

**ERUPTIVE STAGE.** On the third day after the appearance of the initial fever the eruption can be faintly discerned. Its appearance may be postponed until the fourth day; indeed, some observers speak of it as coming regularly at that time. The eruption appears first in the form of macules, scarcely raised above the surface, these rapidly changing to papules. They are first observed on the forehead near the border of the hair, and upon the wrists. The spots are not larger than a millet-seed, and if the eruption is a copious one, the papules are so closely approximated as to cause the eruption to resemble that of measles. Within eight to sixteen hours the eruption appears upon the body and limbs, but is usually scant in these regions. At the close of the first day of the eruptive period the papules have a "shotty feel" beneath the skin. During the second day of the eruption the papules enlarge and vesiculation commences, which process is completed twenty-four hours later. Umbilication of the vesicles now develops, and by the eighth or ninth

day of the disease the vesicle is transformed into a pustule. Then umbilication disappears, the top of the pustule becoming globular and yellowish in color. With the progress of the change in the eruptive points, the skin about the pustules becomes more inflamed, swollen and œdematous. The nose is obstructed by the pustules with their attending inflammation, and the patient finds it necessary to breathe with his mouth open. The throat is sore, the voice husky, and a stridulous cough may be present. Swallowing is difficult. Respiration may be frequent and difficult, and the evidences of broncho-pneumonia present. The rapidity of respiration is generally disproportionately frequent in comparison to the height of the fever and the pulse-rate, so much so at times as to give rise to an apparent dyspnœa. The face often presents a hideous appearance. The eyes are injected, closed, and discharging muco-pus; photophobia may be extreme, and the features not recognizable, owing to the eruption. The hands, feet, and genital organs may present the same swollen, disfigured and offensive condition.

THE STAGE OF SUPPURATION is coincident with return of the fever, and may be preceded or not by a chill, or series of chills. The rise of temperature is rapid, and there is a general return of serious symptoms. The temperature may be even higher than during the initial fever, frequently reaching 106° F., and in extreme cases higher. The subsequent course of the fever is remitting in type, the exacerbations occurring in the evening. If the course of the attack is to be favorable, however, the temperature falls a little each day, the normal being reached in a period ranging from two, three, to eight days, according to the severity of the case.

During the suppurative fever, headache, sleeplessness, pain and often delirium appear. The delirium frequently constitutes a serious symptom, not only in itself, but because the patient is liable to harm himself and others. There may be times in which mechanical restraint is absolutely necessary.

The eruption may, indeed frequently does, appear on the mucous membranes, especially upon those near the orifices. The mucous membranes of the rectum, vagina and urethra generally escape.

STAGE OF DESSICATION. Dessication begins on the eleventh or twelfth day of the disease and occupies from one to two weeks. The pustules rupture, and the contents drying upon the surface, form crusts of various size. When pustulation is extensive, enormous crusts are formed, even constituting moulds of the hands, feet, or portions of the face. With the progress of drying the swelling and redness of the skin gradually diminish, the crusts finally drop, leaving the surface, formerly covered by each crust, of a reddish-brown hue, or somewhat of a cyanotic color with elevated edges. If the ulcerative process has penetrated deeply, a permanent scar is developed, the cicatrix gradually becoming whitish.



If the ulceration has been superficial, a shining white cicatrix results, and this gradually disappears.

The time intervening between the initial development of the disease and the established convalescence is from three to four or more weeks, varying according to the severity of the case.

During the period of dessication intense itching often appears, the desire to scratch becoming irresistible. There is a general belief, founded on error, that it is this scratching which gives rise to the "pitting." The only harm coming from the act is the increase of the inflammatory process, and consequently greater destructive changes. Simple removal of crusts probably exercises no influence upon the pitting.

There is practically no fever during the stage of dessication. When such does occur, it is indicative of some complicating factor, as erysipelas, etc.

The above description represents the prominent features of the ordinary forms of smallpox—*variola discreta*. Occasionally this form is very mild, not all persons being equally susceptible to the influence of the variolous virus, while in others, the pathological conditions and the symptoms are of an aggravated character. When variola is intense in its development, the result is usually a confluent form of eruption, which is attended by constitutional disturbances of a high grade. This variety constitutes the "*variola confluens*."

VARIOLA CONFLUENS. The initial stage of *variola confluens* is always violent. Curschmann says that mild invasion symptoms contraindicate the confluent type. The invasion symptoms are also of shorter duration, because the rash generally appears some hours earlier than in discrete smallpox. Indeed an early eruption should always be looked upon with suspicion even if the invasion symptoms have been mild. The temperature may rapidly attain 106° or 107° F. Even much higher temperatures have been reported. It is seldom that the fall of the temperature and the appearance of the eruption are delayed later than forty-eight or sixty hours after the first symptoms. The special features of the eruption are, the greater distribution of the eruptive points—often appearing simultaneously on the face, extremities, and body—and their close approximation, with the local results which might be expected to follow upon the development of such a closely packed eruption, and such intense inflammation of the skin. Even upon the first day of their appearance the papules may be so thickly developed upon the neck, face and hands, etc., as to touch each other. By the following day, the skin is greatly swollen and vesiculation rapidly develops, followed by coalescence of the vesicles and the rapid development of pus. In this manner the entire face may become an ulcerating surface, and later, covered with enormous crusts. The danger to life from this form of smallpox is very great, one-half to two-thirds of all cases succumbing. The risk to life is judged not only

by the general extent of the eruption, but also by its ravages on the face. If the eruption be confluent on the face only, we must view the case with considerable alarm. Few recover if the body eruption is confluent. As compared with *variola discreta*, it is only necessary to draw an exaggerated picture: Violent invasion, early, profuse, and confluent eruption, which runs a rapid course, great prostration and well-marked typhoid symptoms, especially delirium, greater probability of aggravated pharyngeal and laryngeal symptoms, frequency of broncho-pneumonia, pleurisy, pericarditis, etc., difficulty in taking food, swelling of the parotid gland, vomiting, diarrhœa, and albuminuria, are all conditions which may be present. Death may occur at any stage, and from the general poisoning, even before the development of the eruption. Most of the cases die during dessication. Those who survive are greatly scarred.

VARIOLA HÆMORRHAGICA. In a small percentage of cases of small-pox, blood appears in the eruption, leading to the distinguishing of a special variety of the disease, based solely upon this feature. The presence of blood may be first detected at any stage of the eruption. If it appears in the papules in the first days, the term *purpura variolosa* is applied; if not until the pustules are formed, *variola hæmorrhagica pustulosa*. Blood may appear in the entire eruption or only within limited areas. The earlier it appears, and the more extended its manifestations, the more unfavorable is the prognosis.

PURPURA VARIOLOSA is generally fatal within a few days, often as early as the second day, or if the patient is not overwhelmed by the poison during the first days of the disease, life may be protracted to the fifth or sixth day. The initial symptoms are the same in kind, but more intense in degree than in the ordinary form. The eruption appears early—often by the close of the second day. The papules are dark in color, and punctiform extravasations of blood are developed here and there. The hæmorrhagic spots gradually enlarge, and may be found upon any portion of the body, especially in portions where the vessels are not well supported, as in the conjunctivæ. By the third or fourth day, hæmorrhages from the mucous membranes usually take place. The hæmorrhage into the cutaneous surface may be so extensive as to give a purplish color to much of the surface of the body. The appearance of the patient is sometimes truly horrible. Death may occur before the eruption appears. Delirium and great prostration are early developed in most cases, but the mental sphere is often unaffected, even up to the time of a fatal issue.

VARIOLA HÆMORRHAGICA PUSTULOSA. This variety progresses in every respect as an ordinary active case of *variola discreta* up to the stage of vesiculation or pustulation. The first indication of abnormality of type is the appearance of blood in the eruption. The earlier the blood appears, the more unfavorable the prognosis. Hæmorrhages from

the mucous surfaces occur, but are not as frequent as in the purpuric variety. In women, metrorrhagia, either consequent upon miscarriage, labor, or menstruation, is not at all uncommon. When fatal, as most cases are, death occurs from about the sixth to the ninth day. Scheby-Buck and Osler mention cases of hæmorrhage into the eruption during the vesicular stage, followed by abortion of the eruptions, rapid improvement in all the symptoms, and unusually rapid convalescence.

VARIOLOID, or modified smallpox, is smallpox in an individual who has been previously protected by a preceding attack of the disease or by vaccination. It is characterized by the mildness of its course. The initial phenomena are often severe; indeed, they may be as violent as in the worst cases. One very prominent characteristic of this variety is the remarkable irregularity presented by it. The eruption may appear first upon the most unlooked for portion of the body. It may appear on the second day of the disease, or again be delayed until the fourth. The eruption itself generally runs a rapid and mild course, and causes little or no scarring. Secondary fever is slight or absent.

**Complications.**—Reference to the involvement of the mucous membranes has already been made. The class of ailments thus occasioned forms the principal source of the variolous complications. Thus from involvement of the mouth, Steno's duct becomes affected, and disturbances of the salivary function result; from extension to the Eustachian tubes, middle ear disease and deafness may ensue. The laryngeal complication is often a serious matter, for it may lead to sudden œdema of the glottis and death. Ulcerative changes may also occur in the larynx and extend so deeply as to cause destruction of the cartilages.

Destruction of the eyes by smallpox was formerly more common than at the present time. The vesicles may involve the conjunctiva as already intimated, and even the cornea. Ulceration and perforation may follow. Permanent opacity of the cornea, if not even more serious changes remain.

Other complications of note are bronchitis, pneumonia, cellulitis, erysipelas, localized gangrene, and glandular inflammations.

Complications referred to the nervous system have been rather frequently reported of late years. These partake of the character of meningitis or of multiple cerebro-spinal sclerosis. In some cases neuritis causes a paralysis.

**Diagnosis.**—A study of medical literature would lead one to believe that smallpox is a disease of such plain characteristics as to make it impossible to mistake it for any other affection. This is true so far as the fully developed disease is concerned. Its early recognition is not always easy. Thus it is said that during a certain smallpox epidemic of recent years, a large number of cases were incorrectly diagnosed and sent by physicians to the smallpox hospital; and these cases represented no less



than twenty different diseases. The difficulty must be admitted prior to the appearance of the eruption. Still the high fever and its sudden accession, its association with intense headache and backache, and the presence of other cases of smallpox in the community, and the failure on the part of the patient to have been recently vaccinated, are sufficient data for making a provisional diagnosis—sufficient indeed to isolate the patient, but *not* to send him to the hospital. The presence of the initial rashes has led to a diagnosis of scarlatina or measles.

In those cases of malignant smallpox in which death occurs prior to the development of the eruption, errors are common; but a careful examination of such cases generally reveals the presence of the “shotty feel” of the developing eruption under the skin of the forehead and wrists.

Smallpox sometimes presents quite a similarity to *cerebro-spinal fever*, attended by a purpuric development. Ordinary cases present but little similarity to smallpox; but occasionally, in the absence of any history of exposure or of knowledge of the prevalence of either disease, one is obliged to wait until full development of the disorder before hazarding an opinion.

The most frequent mistakes made are those which confound *measles* with smallpox. In the former affection the eruption appears upon the entire face and the back simultaneously. It is preceded by fever, but that fever is associated with marked catarrhal symptoms. The stage of invasion is less severe in all of its manifestations. The “shotty feel” beneath the skin is absent. The “grissolle sign,” as described by Moore, may be of some service. “If, upon stretching the affected portion of the skin, the papule becomes impalpable to the touch, the eruption is caused by measles; if, on the contrary, the papule is still felt when the skin is made tense, the eruption is the result of smallpox.”

*Chickenpox* has been confounded with smallpox. It is a much milder affection, with decidedly less severe invasion. The varicella eruption appears in crops, and is especially marked over the body. Its vesiculation is more prominent, and the general symptoms are slight.

**Prognosis.**—Before the introduction of vaccination smallpox was the most fatal of epidemic diseases. In recent years very fatal but limited epidemics have occasionally been developed in communities which have neglected to take advantage of this efficient prophylactic. One of the most conspicuous of these was the outbreak in Montreal, Canada, in 1885, the result, as already stated, of the opposition of the French Canadians to vaccination.

The mortality varies much in different epidemics. It is seldom below 20 per cent. in general epidemics. The majority of deaths are among those suffering from variola confluens and the hæmorrhagic variety. The fatality among young children is very great. Little can

be judged of the danger of an attack by the severity of the initial symptoms, but more can be determined by the eruption. Sydenham, whose writings still constitute an authority on this subject, wrote that the extent and intensity of the eruption upon the face and hands afford the best criterion. Strümpell regards the initial erythematous eruption as favorable. Extensive involvement of the larynx, pharynx or lungs adds greatly to the gravity of the patient's condition. Pregnant women seldom survive.

**Treatment.**—On the appearance of smallpox in a household, the first duty of the physician is to attend to the vaccination of every person therein. Exceptions only should be made of those whom *he knows* to have been successfully and recently vaccinated. Concerning the value of vaccinating the patient, there is a difference of opinion. Some writers hold to the view that performed early after exposure (within four days) it will modify or prevent the disease; and in some cases will even modify the attack if delayed still longer. The measure can do no harm under such circumstances, so the patient should be given the benefit of the doubt.

The patient's room should be well aired, and comfortably cool. Under no circumstances should the old notion of shutting the patient up and preventing ingress of air be sanctioned. The diet should be light and liquid, *e. g.*, milk, soups, and broths.

Local antiseptic treatment of the eruption is without positive value. If the pustulation has been sufficiently deep to destroy the true skin, scarring cannot be prevented. If it is deemed advisable to resort to local applications, let it be a very weak solution of mercuric chloride (1:10,000). Other antiseptics in weak solution may be employed.

*Aconite* is a valuable remedy in the stage of invasion. Bæhr, admitting the general favor in which this remedy is held in the treatment of this stage of variola, dissents from such use of it, for, admitting the value of aconite in all inflammatory fevers, he does not think it indicated in febrile conditions that owe their origin to intoxication of the blood. The practical fact of its usefulness is, however, more important than a theory as to why it cannot be of benefit.

*Gelsemium* or *belladonna* may be given if the patient is drowsy, and free from the restlessness, anxiety, and high vascular tension of aconite. *Belladonna* is recommended by Bæhr as the most useful remedy for the stage of invasion.

*Veratrum viride* has manifested a favorable influence if, with high fever, the extremities are cool, the pulse small, and the stomach irritable.

After the eruption has appeared, but little can be gained from the use of these medicines, and they should not be employed unless clear indications for them are present. For the eruptive stage, many remedies have been lauded by different observers. During the first days of the

eruption, while the skin is actively inflaming and the patient complaining of the burning and itching, *rhus toxicodendron* is invaluable, often modifying in considerable degree the cutaneous inflammation and thus favorably influencing the future of the case. The best results have been obtained from the use of the tincture, in doses of from one to three drops every two to four hours. Experience of late years has convinced me of the greater value of material doses of the tincture of this medicine in affections involving the skin. *Apis mellifica* or *croton tiglium* may afford relief, should *rhus* prove inefficient.

The *vaccine virus* internally administered, *tartar emetic*, *sulphur*, and *thuja*, are all represented as possessing a modifying influence over the eruptive stage. *Vaccininum* has been quite extensively used in the treatment of smallpox. Its activity as a drug administered internally has been satisfactorily demonstrated by many observers, including many physicians of the old school. Its efficiency as a remedy for variola has been equally well determined by many clinicians. During the Philadelphia epidemic of 1872, the writer treated many cases, using this medicine in a routine manner, in dilutions ranging from the third to the sixth decimal, giving at the same time, in some cases, a few doses of *belladonna*, *rhus*, or *tartar emetic*, according to indications furnished by special symptoms. The results were quite satisfactory, as among about sixty cases, none died, although many deaths occurred in the same neighborhood. These cases were all treated at their homes, and most were in good circumstances, which accounts largely for the absence of deaths.

*Tartar emetic* enjoys a greater reputation in the treatment of smallpox than does any other medicine. Its close homœopathicity to the disease has been shown by Hughes, and his observations corroborated by many physicians. An "abortive influence over the variolous process" has been claimed for it by Ludlam, Hughes and others. While the single case quoted by Hughes from the writings of Ludlam proves nothing, I am satisfied from some experience with the medicine and the careful study of other experiences, that tartar emetic must be classed among our most valuable remedies. Like *vaccininum* it must be given early, for after vesiculation or suppuration is established, it is of little value, unless sharply indicated. If so indicated, it is generally for a pulmonary or gastro-enteric complication. It is best administered in the third decimal trituration, one grain every one to three hours.

With the appearance of pus in the eruption, *mercurius* should be administered, unless the case is progressing satisfactorily under previously selected treatment. The evidence of its clinical value is overwhelming. Bæhr claims that this remedy, administered on the appearance of the variolous eruption, and at not too frequent intervals, will greatly modify the subsequent suppurative process. From the third to the sixth triturations have been mostly used.



*Baptisia* was brought prominently before the profession as a remedy in variola by Dr. E. Williams, of Bristol, England. He bases his observations upon the treatment of three hundred cases occurring in children in a certain institution. Of seventy-two cases treated with *baptisia*, none died, while of 185 cases treated with the usual remedies, nineteen died. The *baptisia* cases were not selected, and the comparative test seems to have been a fair one.

*Thuya*. The use of this medicine as a preventive and in the treatment of smallpox, was recommended by Bœnninghausen, who claimed most remarkable results in the epidemic of 1849.

In the hæmorrhagic cases, *lachesis*, *crotalus*, *hamamelis*, *phosphorus*, *secale*, *muriatic acid*, and *arsenicum*, are to be thought of.

*Hepar* has been employed to limit the suppurative process.

## VARICELLA.

**Nomenclature.**—Spurious variola; chickenpox.

**Definition.**—An acute, highly contagious disease most frequent in children, and characterized by a vesicular eruption.

**Etiology.**—There are many who endorse the opinion of Hebra to the effect that variola and varicella are products of the same poison; but as an attack of varicella does not confer immunity from variola, and *vice versa*, as epidemics of one may precede or succeed the other, we feel justified in considering it a specific form of disease. Bacteriologists have as yet been unable to describe a specific germ. Nearly all cases occur in young children, *i. e.*, those between the ages of two and five years of age. Varicella occurs in adults much less frequently than do most of the infectious diseases of children. The limitation of the disease to children has been explained on the theory that, from the highly contagious character of the disease, all in any way susceptible to it contract it while yet in early life. It appears, as a rule, in epidemic form. While very contagious, the inoculability of the disease has been called in question. Many experimenters have failed to produce varicella in this way. Some few, on the other hand, have succeeded.

**Clinical Course.**—The specific, indeed the only, lesion is the peculiar eruption. The number of points of eruption may range in individual cases from a few to hundreds. Each point which pursues its normal course passes from a macular to a vesicular stage in from forty-eight to seventy-two hours. The vesicles dry rapidly, and a brown crust is formed by the fifth or sixth day. This crust, on falling, leaves usually a smooth skin, the entire duration of the trouble being from seven to ten days. The vesicles may be either ovoid or globular. Well-developed lesions may be followed by pitting. There are nearly always a few cicatrices that are permanent, and these are often the result of scratching. A characteristic feature of the eruption is its appearance in crops. It is first seen upon the trunk, rarely upon the forehead or face. Its development is usually complete by the close of the third day, so that after this period it may be seen in all its stages of evolution. Observers differ respecting the confluence of the eruption points, some stating its occurrence, while others maintain that it is always discrete. I have witnessed the confluence of two or three vesicles, but never any such bullæ (two inches in diameter) as Loomis and some others have described. Some of the points of eruption may rest upon an inflamed base. There is an absence of umbilication, but some of the vesicles may be divided into

compartments, as in some other eruptive diseases. A scarlatinous rash has been observed to precede the eruption.

The constitutional disturbance is usually slight, but bears a close relationship to the extent of the eruption. The temperature is seldom higher than 101° F., but may continue after the appearance of the eruption. The latter may even appear in the mouth, pharynx, and the mucous membrane of the genitalia.

Several unusual peculiarities have been observed: (1) The development of vesicles into large bullæ. (2) The appearance, as in so many eruptive diseases, of a scarlatinous rash prior to the characteristic eruption. (3) Hæmorrhagic varicella, indicated by cutaneous ecchymoses and hæmorrhages from the mucous membranes. (4) The occurrence of gangrene at the base and periphery of the vesicles. (5) The writer has observed a considerable degree of pharyngitis in connection with the appearance of the eruption in the throat. (6) Nephritis has been exceptionally observed.

**Diagnosis.**—Smallpox is the only disease liable to be confounded with varicella. The appearance of the eruption in the latter in crops, and the absence of the sensation of shot under the skin which characterizes the early stages of the variolous eruption, are strongly suggestive of varicella. There is also later an absence of the infiltrated and reddened base upon which the eruption of variola rests. One is at once impressed with the superficial character of the eruption of varicella as compared with that of variola. The age of the patient has some differential value. Varicella rarely occurs in patients past the twelfth year. Variola may occur at any age. The appearance of the varicella eruption is not usually attended by any marked symptoms; the reverse is the case in variola. The eruption of varicella spends its force on the body; that of variola is general, if not indeed more marked on the face.

**Sequelæ.**—Sequelæ are not frequent, though they may occur. One of the most troublesome is the appearance of sores about the head and body. These are apt to be followed by glandular enlargements and tuberculosis. Other sequelæ reported are nephritis, keratitis, conjunctivitis and vulvitis.

**Prognosis.**—Uncomplicated varicella always ends in recovery.

**Treatment.**—If fever is present or the eruption very profuse, rest in bed is indicated. The diet should be simple. Caution should be exercised to prevent injury to the eruption as it appears on the face. Lint, saturated with a solution of calendula, arnica, or hydrastis, may be applied to the face if there is much inflammation about the base of the eruption points. *Rhus toxicodendron* is the most useful remedy and should be given from the first. *Mercurius*, *vaccininum*, and *tartar emetic*, may also be considered.



## VACCINIA.

**Synonyms.**—Cowpox.

**Definition.**—An acute infectious disease of cows, characterized by the formation of a peculiar eruption upon the udders. From this eruption the vaccine virus is obtained.

**History.**—The existence of cowpox has long been known. For many years it was a so-called superstition that milkmaids who had contracted this disease in the pursuit of their occupation, were exempt from smallpox. Jenner, while yet a medical apprentice, learned of this belief and at once became interested in it. For a period covering twenty years he visited dairies, made inquiries among persons supposed to be acquainted with the facts, only to become convinced that the superstition lay at the foundation of a scientific fact. Finally, on May 14, 1796, he inoculated a healthy boy with the virus taken from the vesicles on the hand of a milkmaid suffering from the cowpox. The patient was subsequently inoculated with the virus of smallpox, but failed to take that disease. Jenner now devoted his life to his great discovery. He visited London and other great medical centres in its interest, but was not received with the enthusiasm the facts warranted. It was his good fortune, however, to live to see himself an acknowledged benefactor of the human race. In 1802 the English Parliament gave him a grant of ten thousand pounds, and two years later twenty thousand more. In 1857 a memorial for all time to come was erected to his memory, it taking the shape of a monument in Trafalgar Square, London. Jenner's theories concerning the nature and action of vaccine were, however, faulty. He taught and believed that one vaccination protected throughout life, and that the disease appeared in the cow by transference from the horse, through the agency of those caring for both animals.

**The Nature of Cowpox.**—Cowpox now and then makes its appearance in dairies through the medium usually of agencies not traceable. The animal first affected is usually a young heifer in its first milk. The lesions appear first upon the teats or the neighboring portions of the milk-bag. They begin as small spots on the third or fourth day after inoculation, and enlarge to form papules and finally vesicles by the fifth or sixth day. Pustulation sets in about the tenth day, at which time there may be some twenty or thirty of these lesions present. The pustules are generally depressed in the centre, that is umbilicated. As in the case of smallpox, the vesicles and pustules do not consist of single sacs, for puncture only empties but one of their compartments. By the

twelfth or fifteenth day crusts form, and these fall off, leaving a scar which persists for years. The clinical course pursued by cowpox is peculiar in one respect. The individual lesions as above described run a short course; but while old lesions are going, new ones come, so that crusts may be seen side by side with fresh vesicles. The whole process may thus be prolonged to cover a period of several months.

The origin of cowpox must be acknowledged a mystery, although very plausible theories have been propounded to account for it. Jenner's idea, as already stated, was that it was conveyed to cows from horses. This was a manifest error, for cowpox appears in localities where horses are not used, and as is quite frequent in Germany, where horsepox is unknown. The generally accepted view is that it is smallpox modified by transmission to a lower animal. All varieties of "pox" may be brought under two classes, those cases occurring in sheep (sheep-pox) and humanized pox or variola. These are held to be identical so far as the poison is concerned. The inoculation of cattle with smallpox virus results in vaccinia, which in its turn produces characteristic vaccination vesicles in the human being. Gassner, of Gunzburg, in 1807, inoculated a cow with smallpox virus from man. There ensued characteristic vesicles, from the contents of which he vaccinated four children with success. French investigators, notably Chauveau, Viennois, and Meynert (and Eichhorst agrees with them), maintain that the two poisons are distinct, and that the cow is incapable of converting variola into vaccinia; that human beings inoculated with the virus thus propagated, develop smallpox. Of late years epidemics of cowpox are more common than smallpox, so that the above explanation appears faulty. The transference of the disease from animal to animal is exceedingly unlikely, for epidemics of cowpox appear at too long intervals to be maintained by continuous succession.

The active agent in the vaccinia virus is undoubtedly of bacterial origin. Ernest and Martin have isolated from bovine lymph a microbe which they have cultivated, and which they claim is capable of producing characteristic vesicles when inoculated in calves and children.

**Method of performing Vaccination.**—The period at which infants should be vaccinated must be selected according to circumstances. When smallpox prevails in a community, no excuse warrants delay, and the operation should be performed at the earliest opportunity. Under other circumstances, it should be postponed until cool weather, and until the child is in good health. The protective influence of a primary vaccination, as the first inoculation with the vaccine virus is called, lasts from seven to ten years, at which time it should be repeated. I would advise repetition at even an earlier date in the face of an epidemic of smallpox in the community, and that irrespective of the general health-standard of the patient.

The virus used should always be that from the cow, as it is readily procurable at the present time. Humanized virus, it is true, takes more easily, but its use is liable to very serious objections. If taken from children the victims of hereditary syphilis, the inoculation of that disease is possible. There has been considerable contention over this point, some authorities claiming that syphilis cannot be communicated through the medium of a vaccine lymph free from blood corpuscles. However that may be, the use of such lymph is entirely unnecessary, and inasmuch as cases are known in which "arm to arm vaccination" has caused syphilis, bovine lymph only should be employed, when such is obtainable. It has been recommended that inasmuch as manifestations of hereditary syphilis appear within the first six months of life, all danger from this source may be obviated by taking virus only from children known to have been in perfect health up to this period. The possibility of conveyance of diseases other than syphilis must also be borne in mind.

The spot selected for introduction of the virus is usually over the insertion of the deltoid muscle, and on the left arm. In the case of female infants, for whom fashion may decree the occasional donning of *decolleté* evening costumes, a scar in this locality may be looked upon with disfavor. Then the operation may be performed upon the outer side of the thigh, immediately above the knee. No special apparatus is necessary for the operation. Care should be observed in every instance to thoroughly cleanse the limb, before proceeding. Many of the accidental complications of vaccination may thus be avoided. Very superficial denudation of the skin should then be made with an ordinary scalpel. Little or no blood should be drawn. The surface denuded should be about one-third of an inch in diameter. All blood should be carefully wiped away; then the virus should be rubbed in from one of the ivory points now in common use. The part should then be exposed to the air until thoroughly dried, after which it may be done up in absorbent cotton, and covered with a shield, while developments are awaited.

Deep introduction of the virus has been advised, but is objectionable, as it is liable to produce furunculosis.

Immediately following vaccination few, if any, local changes are observed. On about the third day slight redness is noted, followed by the appearance of a papule, which on the fifth day undergoes vesiculation. This increases in size and becomes surrounded by a rose-red border, the entire lesion having been not inaptly described by Jenner as "the pearl upon the rose." About the eighth day, the vesicle changes into a pustule; on the twelfth a crust forms, and this drops off several days later, leaving a bluish-red surface, which in turn clears off, leaving a punctated cicatrix which persists for years.



**Complications, Sequelæ, etc.**—Reference to furuncles following vaccination has already been made. Erysipelas has been known to follow this operation, but may be avoided by proper attention to cleanliness and selection of virus. It should be remembered that erysipelas consequent upon vaccination entirely destroys the protective influence of the inoculation.

Vaccination in some instances seems to exert the unfortunate power of rendering dormant constitutional vices active. This is especially true of tuberculosis. Thus have arisen some of the objections to the procedure.

*Vesicular pocks* are occasionally observed. These consist of small vesicles surrounding the initial vesicle, and sometimes covering large surfaces of the body. These form scabs, which drop off without leaving any scar.

*Eczema* is sometimes excited in scrofulous or rachitic children. It manifests itself first by the appearance of minute vesicles about the vaccination, and terminates in an obstinate "weeping" eruption.

*Gangrene* has occurred in children with decidedly depreciated constitutions.

*Roseolar patches* are liable to appear from the third to the eighteenth day. They are unimportant in significance.

**The Treatment** of the unfortunate results of vaccination should be conducted upon the general medical and surgical principles indicated by the condition present. *Silica*, *thuja* and other remedies have been specifically recommended, but their use savors of empiricism. Erysipelas, tuberculosis, vaccino-syphilis, eczema, etc., must all be treated according to the individuality of the case.

Secondary vaccinations do not follow the course of the primary cases.

**Inoculation.**—Inoculation being the measure which preceded vaccination as a preventive of smallpox, a few words concerning it in this case may not be inappropriate. According to many authorities inoculation was practised in China for thousands of years prior to its introduction into Europe. According to others, the Chinese are so stupid and ignorant of medical matters as to have known nothing about this or any other genuine remedial agent. However, it was introduced into Europe by Lady Montagu, the wife of the British Ambassador in Constantinople. The first experiments were performed upon three criminals condemned to death, and who were given their liberty in return for their affording the opportunity of experiment. It was first performed in America in 1721 at Boston. The disease thus produced fully protected the patient from subsequent seizures of variola, but it was open to the very serious objection that it communicated smallpox to others who perchance came in contact with the patient. While the artificial disease was much lighter

than that naturally produced, it was not devoid of danger, deaths resulting in from one out of eighteen cases (Ferro) to one out of six hundred and sixty-two cases (Wilson). The skill of the operator had much to do with the result, the minimum amount of virus capable of producing the artificial variola being the proper dosage.

## WHOOPING COUGH.

**Nomenclature.**—Pertussis; tussis convulsiva.

**Definition.**—A specific, contagious disease, characterized by paroxysms of coughing, attended at their close by a long-drawn inspiration, to which the name of “whoop” has been applied, and from which the disease has derived its name.

**Etiology.**—The single cause of whooping cough is exposure to the contagium of the disease. The nature of the infectious agent cannot, as yet, be said to have been positively established. Still, Afanassiew has discovered a bacillus which is invariably present, and which, when injected into the trachea of the lower animals, has produced a catarrhal condition of the mucous membranes. Smischenko has confirmed this observation respecting the whooping cough bacillus.

Age exerts a great influence respecting the susceptibility to the whooping cough contagium. Children at about the age of two years appear to be the most susceptible; at the age of six, susceptibility diminishes, and by the tenth year, it is almost extinct. Adults may take the disease; but with them it almost always runs a mild course.

The degree of contagiousness of this disease is not believed by the majority of clinicians to be great; consequently, it is held that a slight degree of isolation only is required to prevent its spread. The evidence of spread by means of fomites is slight. Transmission from patient to patient is the rule. Eichhorst dissents from these views, and considers whooping cough highly contagious, has firm faith in its transmission through third parties and by fomites generally, and strongly urges the strictest isolation.

The contagious principle is believed to reside in the expectoration, and possibly also in the expired air. Eichhorst accounts for much of the spread of the disease to the presence of powdered sputum in the atmosphere.

Whooping cough may occur in epidemic or sporadic form. The latter cases, it must be remembered, arise by infection in some way. Epidemics are especially liable to occur in the winter and spring months. They very frequently appear when measles is prevalent in the community. Patients suffering from the latter disease, as well as women during pregnancy and in the lying-in period, are especially susceptible to it.

One attack of the disease generally renders the patient immune to subsequent ones.

**Pathology and Morbid Anatomy.**—There are no essential lesions



in whooping cough. Catarrh of the respiratory mucous membranes is often present; but it does not present individual peculiarities.

**Symptoms.**—The clinical course pursued by whooping cough is characterized by two stages, the first of which, the catarrhal, differs in no essential particular from the ordinary catarrhs of the respiratory mucous membrane, and which merges into the second stage, the paroxysmal, in which the peculiar paroxysmal cough is a prominent feature. The stage of incubation is said by different authorities to range between seven and fourteen days.

The catarrhal stage lasts generally from a week to ten days. As already intimated, it begins like an ordinary cold with coryza, conjunctival injection, lachrymation, dry bronchial cough, and some degree of fever, especially in the evening. The possibility of whooping cough is foreshadowed by the unusual amount of prostration attending these symptoms, and the paroxysmal occurrence and nightly aggravation of the cough.

The merging of the two stages may be sudden, or by almost imperceptible gradations. In the spasmodic or paroxysmal stage, the attack consists of a series of expiratory coughs, terminating by a long-drawn, loud inspiration—the whoop. The attacks are preceded by a premonition or aura, consisting of a vague sensation, sense of oppression, dyspnoea, or impending danger, and causing the little sufferer to leave its play, and run to its attendants. The prolonged duration of the paroxysms and the expiratory character of the cough make phenomena attendant upon deficient aëration of the blood prominent features. As the attacks may last from one-half minute to five minutes, and as but little air passes the glottis during this time, it is not to be wondered at that the face becomes swollen and turgid, that the bloodvessels of the face and neck stand out like whip-cords, that the conjunctivæ become injected, and that suffocation appears imminent. Then the deep crowing inspiration takes place, there is vomiting or expectoration of a thick, glairy mucus, the attack is over, and the cyanotic symptoms at once disappear. The frequency of the paroxysms is a pretty reliable index of the severity of the disease. In exceptionally mild cases, there may be but three or four of them in the course of the twenty-four hours; while in severe cases, they may attain as high a number as from fifty to one hundred in the same time. They are almost always worse at night.

The very violent character of the cough produces certain hæmorrhagic symptoms, prominent among which are epistaxis and subconjunctival ecchymoses. Hæmorrhage may very exceptionally take place within the cranium, or in other portions of the body. The severe congestion of the head may produce quite severe vertigo, and Sebergundi has reported a case in which there was temporary loss of sight during the paroxysms. General convulsions even may result, not very frequently it is true, but they may nevertheless constitute a dangerous symptom.

Sometimes the expectoration is attended by vomiting, and may be streaked with blood. Albuminuria has been reported by Steffen.

During the intervals between the attacks the patient may present a typically healthy appearance. Physical examination at such times reveals nothing excepting a few bronchial râles, altogether insufficient to account for the severity of the case. In probably 40 per cent. of the cases an ulcer will be found beneath the frænum of the tongue. For a long time this ulcer was believed to be the cause of the peculiar cough, until it was learned that it only appeared in children after dentition, and was the result of the friction of the tongue against the incisor teeth during the paroxysms. After the spasmodic stage has lasted from two to four weeks, amelioration of the symptoms begins. The intervals between the paroxysms increase, and the attacks themselves grow less severe. The course of the disease may, however, be quite protracted.

**Complications, Sequelæ, etc.**—Hæmorrhagic symptoms may be prominent, petechiæ appearing in various parts of the body. Even hæmoptysis and subdural hæmorrhage may ensue.

Vomiting, so often a mild symptom, sometimes assumes such a serious phase as to affect nutrition, and lead to fears of death from exhaustion, or at least induces anæmia and emaciation.

The most serious complications of whooping cough, and the ones, moreover, which most frequently occasion a fatal result, are those involving the respiratory tract. Bronchitis is by no means uncommon, and broncho-pneumonia with attendant collapse of the air-vesicles is often observed. Pleurisy and lobar pneumonia likewise occur. Pulmonary emphysema is a rather frequent attendant, although it does not occur with anything like the frequency one would expect from the degree of strain to which the lung tissues are subjected. In any event it is always slight in degree, with the exception of cases in which dyscrasic conditions exist.

Like measles, whooping cough seems to have the power of rendering a latent tuberculosis active.

Occasionally nervous complications have been noted; of especial importance in this connection are convulsions, depression of spirits, and paralysis.

**Diagnosis.**—The recognition of the disease during the catarrhal stage is important, but can only be made by a knowledge of the exposure of the patient to the contagium of whooping cough, and the fact that he is of a susceptible age. The nightly aggravation, the paroxysmal nature of the coughs, and the disproportionately mild physical signs, are of course strongly suggestive. Trousseau was accustomed, during this period, to lay great stress on the incessant character of the cough. During the spasmodic stage, the recognition of the disease is an easy matter.

**Prognosis.**—Whooping cough is generally recognized as a mild dis-

ease, and with good reason, for very few cases free from complications and properly treated die. The general mortality is said to be from  $\frac{1}{2}$  to  $1\frac{1}{2}$  per cent. Carelessness in its management, its occurrence in debilitated and anæmic subjects, have made it at times a very fatal disease. In England it ranks third in the mortality list among children. There have been decades in which whooping cough has, in New York City, caused more deaths than typhoid fever. The danger seems to be much greater as the subject is younger. As high a death-rate as 48 per cent. has been reported among subjects in the second year of life. Adults have the disease very mildly. The prognosis is affected by the number of daily attacks. Thus when these reach over fifty in twenty-four hours, there is considerable cause for anxiety.

The prognosis of broncho-pneumonia complicating whooping cough is unfavorable, nearly one-half of the cases dying. This statement is based upon old school authorities.

The danger of subsequent tuberculosis must not be forgotten.

**Treatment.**—PROPHYLAXIS. It is wise to remove delicate children from an infected neighborhood. If they must remain, all communication or association with the infected should be avoided. The period during which one may contract the disease from a sufferer has not been accurately determined, but some say it is infectious as long as the cough is present. This is perhaps a good general rule, but cannot be depended upon as representing the exact truth. Recrudescences, even if associated with a well-defined whoop, are considered free from danger to others.

GENERAL CARE. An abundance of fresh air usually diminishes the number and intensity of the paroxysms. Wool should be worn next the skin, and exposure to cold and damp air avoided. When the weather permits, exercise in the open air should be taken daily. The nutrition should not be neglected. Nutritive enemata may even be useful, especially if vomiting is excessive. Changes of climate which can be made with ease result in benefit, especially in protracted cases.

MEDICINES. There are few diseases in which a larger number of remedies have been recommended. This implies unsatisfactory results. In making estimates of the value of medicinal agents, one must consider the epidemic, the stage of disease when administered, etc. In the catarrhal stage the treatment is the same as for ordinary bronchitis, at least it must be so, as the nature of the disease is often not suspected. At this time *aconite*, *belladonna*, *ipécacuanha*, and *hyoscyamus* are the main remedies. If the child has been exposed or an epidemic is present, remedies of known value in whooping cough should be selected. I have, in recent years, had the most satisfactory results from the use of *naphthalin*, which I have given as soon as the nature of the case has been apparent. The first decimal trituration has been most generally useful. It should be frequently repeated. Other medicines—such as *belladonna*—may, if indicated, be



given for a time without discontinuing the *naphthalin*. The old Hahne-mannian remedy, *drosera*, is often valuable in the spasmodic stage. It may succeed in doses of five drops of the tincture when dilutions fail. The same may be said of *mephitis*. I have seen several cases in which the most violent symptoms of whooping cough have been relieved in so short a period of time as to permit of no other conclusion than that the relief was due to the action of this medicine. The spasmodic element and the vomiting were pronounced. *Cuprum aceticum* is recommended by many. It has proven only occasionally useful in my experience, but then in most serious cases, viz., in neurotic children with most violent paroxysms, convulsive action of muscles, or general convulsions, cyanosis, numerous râles in the chest, and albuminuria. Jousset recommends *cina* and *coccus cacti*. The former is often useful when given for its well-known indications. *Corallium rubrum* (Teste) enjoys some reputation for the spasmodic stage. I have had little experience with it. Hale reports good results in a recent epidemic from the *bromide of gold*, second decimal trituration, two grains daily. I have had some favorable experience with *aurum muriaticum*, but insufficient for conclusions. In the writer's experience very great assistance has been secured from the use of inhalations as recommended for pulmonary phthisis. They should be repeated three or four times in the twenty-four hours. Each inhalation should last one hour. Shorter periods may be employed, until the child becomes accustomed to the method. The best results have apparently followed the inhalation of *beechwood creosote* and *terebene*. Quite remarkable results have been of late secured through the fumigation of the patient's night-room by means of burning sulphur. The child is to be removed from the room in the morning, washed and dressed in clean, fumigated clothing. At the same time the closed sleeping-room is to be fumigated for five or six hours by burning sulphur. One application of this method has proven successful. Insufflations of *resorcin*, one-half to one grain being blown into the nose several times daily; or a one per cent. solution applied to the pharynx and larynx as far as possible, are recent suggestions. *Bromides*, *morphine*, *benzoin*, *peroxide of hydrogen*, and a variety of agents are recommended for local use in the form of sprays and insufflations. They are not much used, and I do not think they are likely to be. *Opiates*, *bromides*, *chloral*, *belladonna*, *hyoscyamus*, *cannabis indica*, *antipyrin*, *phenacetin*, and other powerful drugs are much employed in large doses by many practitioners for the control of the spasmodic stage, but must be rarely necessary. *Bromoform* as a remedy for whooping cough was introduced in 1887 by Stepp, and has become quite popular. Doses of two to four drops according to the age of the patient are advised to be given every four to six hours. It may be administered upon sugar or in water. My experience with this remedy is moderate and not very encouraging.

COMPLICATIONS. The most important ones have been seen to involve the lungs and brain. In the treatment of the former, *phosphorus*, *tartar emetic*, *arsenate of antimony*, *arsenic*, *sulphur* and inhalations of oxygen are valuable remedies. For the nervous symptoms, *opium*, *cuprum*, *bella-donna*, *hyoscyamus*, etc., may be considered, but remedies directed to the cough, and any attending pulmonary complications, are of vastly more importance, since the nervous phenomena are generally the result of these. For the details of treatment of the various affections included under these headings, also for the treatment of hæmorrhage, vomiting and other troublesome symptoms, see the appropriate sections. Protracted convalescence is best treated by change of air and persistent inhalations, according to method already referred to.

## EPIDEMIC PAROTITIS.

**Nomenclature.**—Mumps; parotiditis.

**Definition.**—Parotitis is an inflammation of the parotid gland, which occurs not only as a secondary complicating condition in a variety of serious affections, but as an acute infectious disease, which becomes at times epidemic. The following description refers to the latter variety exclusively.

**History.**—Mumps has been known from the most remote ages. Hippocrates presented very good descriptions of it in his writings. For many years, epidemics of this disease have been known to occur in association with epidemics of other infectious disorders. The relationship between these troubles is not, however, understood.

**Etiology.**—There can be no doubt of the specific nature of mumps, although we do not yet know anything of the nature of the specific sort of virus which, it is presumed, makes its way into the glands *via* Steno's duct. Its germ nature has of course been suggested, and the *bacillus parotiditis* said to have been isolated. The element of contagion is believed by some to reside in the saliva. This being expectorated by those infected, dries and permeates the atmosphere, thus communicating the disease to others. There is some evidence to show that third parties may convey the disease. The period during which patients are infectious is believed to range from one day before the appearance of the glandular enlargement to two weeks after the disappearance of febrile symptoms. Whether or not cases still affected with the so-called metastatic inflammations are dangerous to the well, is as yet unknown.

The disease is apt to appear in epidemics, which may be quite extensive. It is endemic in most cities. Spring and fall seem to be the periods of time most favorable to its spread. Infants and the aged are seldom attacked. Children and adolescents are most susceptible to it. Males are more frequently affected than females.

**Pathology and Morbid Anatomy.**—There is some diversity of opinion among pathologists respecting the location of the earliest morbid changes. This may be due to the infrequent opportunities for post-mortem examinations. It seems probable, however, that the primary changes occur in the parotid gland, and are of the nature of a catarrh of the ducts. The gland becomes greatly swollen and presents on post-mortem examination a fleshy appearance; but much of the swelling which imparts to this disease its peculiar appearance is due to involvement of the peri-glandular connective tissue. Suppuration is rare. Permanent



enlargement with induration and atrophy of the gland have been observed as unusual sequelæ.

**Symptoms.**—After a period of incubation averaging about two weeks fever appears. This may be followed at once by pain or swelling, or both, below the ear upon one side. This swelling rapidly increases and within two days there is often great enlargement of the neck and neighboring cheek. To this may be added a swelling of the floor of the mouth. Both parotid glands are usually affected, but the involvement of one generally precedes that of the other by two, three or more days. The degree of swelling varies much, at times it involves the gland mainly; at others, the neck and cheek are enormously swollen, and if both sides are affected the appearance of the patient is wonderfully changed. Owing to the stiffness and pain resulting from motion, the head is held in a fixed position, rotation being difficult. The skin over the swelling may be pale, or glossy and red. Mastication and swallowing are so painful that but little food is taken. Movements of the jaw, as in speaking, also give rise to much suffering; the patient talks little and the voice is unnatural. In some instances the pharyngeal mucous membrane may manifest some degree of œdema. The saliva may be much increased, but even if this is not the case, it may dribble from the unclosed mouth. Pain in the ear is quite common. Varying degrees of impairment of hearing may exist temporarily or permanently. The duration of typical cases is from a week to ten days, at which time diminution of the swelling takes place, with a simultaneous relief of all symptoms.

**Complications.**—Perhaps the most striking peculiarity of mumps is the tendency to involvement in a limited number of cases of the sexual organs of both the male and the female. Orchitis is most frequent. It develops usually with the decline of the swelling of the parotid. One or both testes may be affected; effusion into the tunica vaginalis is frequent. Suppuration is a rare sequence, and the same may be said of atrophy. The tendency to a complicating orchitis is much greater after adolescence. Bruising of the testes has been said to favor the development of orchitis, and the writer has observed a case seemingly due to sexual intercourse during the progress of the mumps.

The testicle is usually not affected until after the swelling of the parotid gland has subsided. In very exceptional cases, the orchitis may be the sole evidence of the disease. The frequency of this complication varies greatly in different epidemics. In some, but one out of a hundred cases is thus affected; while in others, fully one-quarter of the patients are sufferers from this complication. One writer refers to an epidemic in which orchitis was the rule rather than the exception. Another observer reports cases in which metastasis to the liver, lungs, and other viscera occurred. In the case of a pregnant woman the

uterus was affected, and abortion and death resulted. Facial paralysis has been exceptionally noted. It is strange that this complication has not been referred to more frequently. Schmidt observed a case in which a discharge from the urethra appeared and disappeared with the glandular enlargement. Acute affections of the eyes and ears appear to be occasionally encountered. The nervous system even may suffer. Meningitis has been reported, and Dr. Percy Smith instances two cases of insanity following immediately upon attacks of mumps. Both patients had been greatly debilitated, one by diarrhœa, and the other by orchitis.

In garrisons especially is orchitis frequent. Jarvis reports an epidemic at Fort Apache, Arizona, during which one-third of those affected had orchitis. Of thirteen cases of the latter, three resulted in atrophy. In women the mammæ or ovaries may be attacked, and in young girls a vulvo-vaginitis has been frequently observed.

In certain epidemics all of the salivary glands have been swollen, and in others the disease has attacked one gland only, and that not the parotid. Some years since an epidemic of mumps occurred in Erlangen in which the submaxillary gland only was attacked in many cases.

**Diagnosis.**—The diagnosis of mumps is not generally attended with difficulty. An acute swelling in the region of the parotid gland, which raises the lobe of the ear, and which is not associated with or a sequence of other diseases, is, in all probability, mumps. The presence of an epidemic, the mildness of attending constitutional symptoms, and the rapid disappearance of the swelling, are all likewise suggestive. In the rare cases in which more than one gland is involved, or in cases in which the parotid is not attacked, doubt must exist for a time at least.

**Prognosis.**—In the vast majority of cases, mumps pursues a mild course, ending in recovery, and this, even without treatment. The occasional orchitis and less frequent atrophy of the testicles or occlusion of the spermatic duct which results, also the possible deafness or eye troubles, must not be forgotten. During the height of the attack the occurrence of suppuration, high temperature, meningitis, delirium, etc., must also be considered. In diseases in which, as in the one under consideration, danger is trifling, and serious complications rare, the physician is apt to be caught napping.

The occasional serious consequences of the orchitis must be borne in mind. Jarvis's observations have already been referred to. Even more serious results have been reported by Cornby, who found that among the troops 103 out of 163 cases of orchitis were followed by atrophy.

**Treatment.**—It is wise, even in mild cases, to confine the patient to his room. If the attack is severe, or fever present, he should remain in bed. The food should be light, nutritious, and, of necessity, liquid. Some comfort may be secured by protection of the inflamed area from the air by some light form of dressing. Hot or cold water dressings are

advised by some. If the age of the patient favors metastasis to the sexual organs, extra precautions in the general care of the patient should be observed in order to prevent such a result.

The support of the testicles by a properly adjusted suspensory is a wise precaution. In case of an orchitis, rest must be absolute. If the inflammation has been severe, faradic applications, after active inflammation has subsided, are recommended as a means of preventing atrophy of the organ.

Hale indicates his preference for local treatment in the shape of application of *belladonna* or *phytolacca* ointment, using the same remedies internally. Medicinal and hygienic treatment, however, will be found sufficient in nearly all cases.

If febrile symptoms are present, *aconite* or *belladonna* will generally be found sufficient. If these or other medicines are not clearly indicated *mercurius* had better be administered, and at any rate if the swelling is marked. *Rhus tox.* is equally valuable, especially if there is extensive œdematous swelling. Threatened suppuration calls for *hepar* or *sulphur*. This remedy or *sulphur* is helpful if resolution does not proceed normally; for persistent induration, *conium*, and *baryta iod.* For the complicating orchitis or mastitis, *pulsatilla* is generally sufficient. *Clematis* has also given good results. Cerebral complications call for *belladonna*, *cuprum aceticum*, *hyoscyamus*, etc. For the treatment of the other complications, the sections treating of the various diseases liable to occur in association with mumps should be consulted.



## TETANUS.

**Synonyms.**—Trismus; lockjaw.

**Definition.**—An infectious disease, produced by the inoculation with the bacillus of tetanus, characterized by paroxysms of tonic spasm of the entire body, and rapidly tending, in the majority of cases, to a fatal termination.

**History.**—Tetanus has been recognized and very accurately described from remote ages. Aretæus and Hippocrates both furnished us with very graphic accounts of its causes and phenomena. Recent years have added but little to our knowledge concerning it, until 1885, when Nikolaier discovered in the earth a bacillus, which, inoculated in the lower animals, produced all the phenomena of tetanus. Rossbach, in the following year, discovered the same bacillus in a case of human tetanus. In 1887, Briege isolated a number of toxins from pure cultures of the tetanus bacillus. These are known as tetanine, tetanotoxine, and spasmatoxine. The injection of these substances into the blood produces all the symptoms of tetanus.

**Etiology.**—The study of the causes of tetanus was always an interesting one. It is no less so since the discovery of a specific bacillus, the inoculation of the system by which is necessary to the production of the disease. This bacillus, as already stated, is found in vast numbers in the ground nearly everywhere, but especially in cultivated districts, and in cities. It is also wafted about in the dust, and is found in the dust of walls and floors. With such an omnipresent bacillus one would expect tetanus to be a very common disease. Such is not the case, however, and probably because of the ease with which the bacillus of tetanus is destroyed by exposure to the atmosphere. The bacilli themselves are thus rendered innocuous before taking up their abode in the human body; and, even if they should be inoculated while in a still active state, they must penetrate the tissues sufficiently deeply to escape destruction by the oxygen of the air.

The bacillus of tetanus consists of a fine rod, a little longer than the bacillus of mouse septicæmia. It may form into threads. Usually, however, and this is its characteristic feature, one end of it is surmounted by a spore, thus giving the structure an appearance that has been described as that of a pin; its appearance as to shape is more like that of a gimp-tack. The bacilli are easily colored with methyl-blue and fuchsin.

The great majority of traumatic cases occur in males. The dis-

covery of this bacillus, and its toxines, and their effects on being introduced into the system, has cleared up much of the mystery surrounding the production of tetanus by slight wounds.

It has long been customary to divide tetanus etiologically into traumatic, rheumatic and puerperal. A variety has been described as occurring shortly after birth and given the special designation of tetanus neonatorum. It is very common among the negro children of the West Indies.

Age, race and sex act as predisposing factors in the etiology of tetanus. The largest number of cases is found in subjects of from ten to twenty years of age. The disease is comparatively rare after forty and before the fifth year of life, excluding, of course, cases of tetanus neonatorum. Men are far more frequently affected than women, in the proportion of six to one, and, in all probability, because they are more exposed to injury and to the vicissitudes of weather. Negroes and, indeed, all the dark-skinned races, are far more predisposed to the disease than are the lighter ones. It is more than probable that these people, full, as they are, of superstition, treat their minor injuries according to the traditional methods of their neighborhood, making all sorts of septic applications, thus inoculating themselves with the tetanus bacillus.

Locality and climate favor the production of tetanus. Thus the disease was for a long time very prevalent on the eastern shores of Long Island and in tropical countries generally. In the West Indies it is, as already stated, very common among the negro children, and is a greater source of mortality than all other infantile diseases combined.

The exciting cause of tetanus is, in nearly all cases, an injury. The wound may be of any character. Usually, however, it is very insignificant, and may be described as a punctured or contused wound. Incised wounds, especially after surgical operations, very rarely lead to the disease. Tetanus has frequently followed the stings of bees and wasps, the prick or scratch of a splinter, the extraction of a tooth, and other very slight traumatisms. In the light of the discovery of the tetanus bacillus and its toxines, of the knowledge of the manner in which the injury is received, and of the common neglect or pernicious treatment of slight injuries, the production of the disease becomes obvious. Punctured wounds producing tetanus are almost always caused by a rusty nail or a dirty splinter. In rural districts it is very common to make applications of "rich" mud (being generally a mixture of stagnant water and very dirty earth) as a remedy for bee and wasp stings. Tetanus has been reported as occurring after injuries which involved no solution of cutaneous continuity. Such cases were reported before the discovery of the tetanus bacillus; it is more than probable, therefore, that they were insufficiently investigated.

Puerperal tetanus and tetanus neonatorum must be regarded as varieties of traumatic tetanus; in both, infection takes place through abraded surfaces. Puerperal tetanus is especially liable to occur after abortions. It was formerly claimed that the disease bore an important relation to laceration of the cervix. It has been observed to occur especially in cases in which tamponade of the vagina has been practised. Tetanus neonatorum was ascribed by Marion Sims, and still later by Hartigan, to displacement of the occipital bone by long continuance of the infant in faulty positions. Displacement of the occipital bone undoubtedly produces tetanoid symptoms, but not tetanus. This disease in the infant is nearly always the result of infection *via* the umbilicus.

**Pathology and Morbid Anatomy.**—Practically nothing is known concerning the morbid changes of tetanus; no definite alterations in the tissues being discoverable post-mortem.

**Clinical Course.**—Tetanus presents an extremely variable stage of incubation. In the majority of cases it ranges from ten to fifteen days. It may, however, be much longer or much shorter than this period. Many times symptoms have appeared within two or three days after the injury, and one case even has been reported in which the patient, a negro, was dead of tetanus within fifteen minutes after cutting his hand on the edge of a broken saucer. In other cases, tetanic symptoms may be delayed until three weeks or more after the accident. When the disease follows abortion, there is usually an interval of from five to thirteen days.

Occasionally the invasion of tetanus is marked by prodromic symptoms, consisting of headache, epigastric distress, and pain at the seat of the wound. Usually its first manifestation consists of a stiffness of the muscles of the neck or jaw or both. This stiffness rapidly increases in degree until it amounts to absolute rigidity. The tonic spasm thus inaugurated spreads rapidly from above downward, involving first the muscles of the spine and lastly those of the extremities, until finally the entire body is rigid, a condition receiving the designation of orthotonos.

The appearance of the face is characteristic. While all of the muscles are affected, those that are strongest impress their actions on the features. Thus the eyebrows are drawn upward by the occipito frontalis, and the palpebral fissure is lessened by the contractions of the orbicularis palpebrarum. The angles of the mouth are drawn outward by the muscles attached to the orbicularis oris, and the upper lip is drawn tightly against the teeth. Thus we have the appearance that has been for ages so graphically described as the risus sardonicus.

The general condition of tonic spasm is from time to time interrupted by spasms, which consist of an increased rigidity, lasting for but a



few seconds. These are often accompanied by cramp-like pains. During their continuance the body is usually arched backward so as to rest on the head and heels (opisthotonos). Very exceptionally, indeed, the spasm of the muscles on the anterior surface of the body predominates, and the arching is then forward (emprosthotonos); or the muscles of the side are involved, and the distortion is to one side (pleurosthotonos). The spasms are greatly intensified by the slightest external impression.

The respiratory muscles are usually affected in the spasms. This is displayed by the short, quick breathing. During the spasm the glottis may be closed, and asphyxia sometimes result. It is believed that the diaphragm is also involved, thereby producing a most distressing epigastric pain, which is intensified during the spasms. The temperature may or may not be raised. At times it ranges high, the increase going on even after death and reaching the very high point of  $110^{\circ}$  or  $114^{\circ}$  F.

It is customary with some authors to describe an acute and a chronic tetanus. This is entirely unnecessary, inasmuch as all cases that recover must pass through a chronic stage.

**Diagnosis.**—Tetanus can only be confounded with strychnia poisoning, tetany, and hysteria. Only the most culpable carelessness should lead to mistaking it for inability to open the mouth from local inflammatory conditions. From strychnia poisoning it is differentiated by the history of the case, the knowledge of a wound, and the period of incubation. Tetanus begins with trismus, which is a late symptom of strychnia poisoning; the latter begins with gastric disorder and the spasm starts in the extremities. In tetanus the rigidity is constant; in strychnia poisoning there is relaxation between the paroxysms. Strychnia poisoning soon ends in death or recovery; tetanus is more prolonged.

The differentiation from tetany will be found in the chapter devoted to that disease.

To distinguish tetanus from hysteria one must rely upon the attending circumstances. In the latter disease there will always be some symptom or condition serving as a very reliable guide-post.

**Prognosis.**—Tetanus is a very serious disease, fully four-fifths of the traumatic cases dying. Those resulting from exposure offer a mortality of 50 per cent. In individual cases one can only state the prospects by the length of the stage of incubation, the longer the period intervening between the reception of the wound and the onset of symptoms, the more likely is recovery to ensue. When fatal, death usually takes place in from three to seven days. The cause of death may be respiratory spasm, heart-failure, or exhaustion from the long-continued spasm. If the patient is able to sleep, the outlook is better than were it otherwise. Signs of improvement are lessening of the number of spasms and increased ability to sleep.

**Treatment.**—By way of prophylaxis, all wounds should be treated on strictly antiseptic principles. All dirty local applications, mud and manure poultices, bandaging with soiled linen, etc., must be avoided.

When the disease has once appeared, amputation of the wounded part is utterly useless, orthodox practice though it was but a few years ago. The most important therapeutic agent for the tetanus patient is *rest*. This rest must be as absolutely perfect as human ingenuity can make it. Every special sense should be blunted to the external world. The room should be darkened, the ears plugged with cotton, drafts of air excluded, and the attendant should so conduct herself as to make no noise whatever in the performance of her duties. Nourishment must be liquid, owing to the locked condition of the jaws.

Antispasmodic treatment is in vogue among the old school, enormously large quantities of bromide of potassium and chloral being prescribed at comparatively short intervals.

Collis Browne's *chlorodyne*, which owes its efficiency to the cannabis indica it contains, is invaluable in some cases.

*Strychnia* is the leading homœopathic remedy in tetanus. Testimony of its efficiency has been given by prominent physicians of both schools of practice. Stille and Trousseau both praise it highly.

*Aconite* can also produce spasms closely simulating those of tetanus. It causes trismus, stiffness of the limbs, and opisthotonos. It is indicated in cases with more or less fever, in which numbness and tingling are prominent subjective symptoms, and exposure to cold has operated with traumatism as a cause. Reynol used this remedy successfully for the trismus of horses, and it was at one time the main reliance in the treatment of tetanus in the Middlesex Hospital, London.

*Hydrocyanic acid* is highly praised by Hughes. It is adapted to cases in which the reflex excitability is much less than in the strychnia patient. Tonic spasm is persistent.

*Passiflora incarnata* is much used in the West Indies as a remedy. Two cases cured by it were reported by Archibald Bayne in the *Hahnemannian Monthly* for May, 1881.

Other remedies, worthy of consideration and study, are *cicuta*, *nicotinum*, *veratrum viride*, *hypericum*, *belladonna*, *curare*, *physostigma*, and *picrotoxin*.

Within recent years, investigations looking to the treatment of tetanus by a serum from immunized animals and toxines have been carried on by Behring, Kitasato, and Tizzoni. While favorable results have apparently been secured in the treatment of tetanus in animals, success can hardly be said to have been secured in the case of the prevention or cure of the disease as it affects human beings.

## ANTHRAX.

**Nomenclature.**—*Carbunculus contagiosus*; malignant pustule; charbon; splenic fever (because of the regularity with which the spleen is involved); the Germans give it the name of Milzbrand, or inflammatory death of the spleen; the Russians call it jaswa or boil-plague.

**Definition.**—Anthrax is an acute contagious disease caused by the bacillus anthracis, and characterized by the appearance of a boil with a black necrotic centre, with infiltration of the surrounding cellular tissue, and followed by systemic infection.

**History.**—Anthrax is primarily a disease of the lower animals, especially those of herbivorous habits. Affecting them, it was known from remote times. The ancient Romans spoke of it as carbunculus. The Arabians are believed to have recognized its existence in man. Many and varied were the ideas held concerning its nature. Some believed that it resulted from over-nourishment of the animals affected; others, that it arose from peculiarities in the soil; and still others that it was purely a malarial neurosis. Positive data proving the existence of the disease in man, were not presented until the latter part of the eighteenth century. The bacillus of anthrax was discovered by Pollender, in 1849, but that observer did not make his investigations public until 1855. Two years later, acting independently of Pollender, Brauell confirmed that author's observation. These bacilli were not recognized as such until 1863, when they were so pronounced by Davaine. Inasmuch as they were motionless, he called them bacteridiæ.

**Etiology.**—Anthrax can arise only from infection. This accident may occur in one of several ways: (1) By inoculation of wounds or scratches while handling carcasses or parts of animals dead from anthrax. In this way butchers, tanners, upholsterers, wool-sorters, shepherds, and veterinary surgeons, become infected. (2) It has been proven that the disease can be conveyed by the bites of insects that have been feeding upon carcasses of animals dying of anthrax. Microscopic investigations have revealed the presence of the bacillus anthracis in the stomachs of several insects; and inoculation of susceptible animals with various portions of these insects has produced anthrax. (3) The disease may arise from eating raw or partially cooked meat obtained from carbunculous animals. In the majority of cases, the flesh of diseased animals may be eaten with impunity. The bacilli are readily destroyed by the gastric juice. If, however, the anthrax spores are present, internal infection follows. Thorough cooking, of course, removes all danger.



(4) Inhalation of dust from parts of diseased animals produces the thoracic variety of anthrax. This is especially liable to occur in wool-sorters; and hence has been called wool-sorters' disease.

A few words concerning the origin of the disease in animals. It is on rare occasions only that the disease spreads directly from one animal to another. Almost invariably they contract it from the soil. When once a given region becomes infected, and this is especially liable to occur from superficial burial of dead animals, that place thereafter becomes a centre of infection from which it is next to impossible to drive away the disease. The reason for this will be explained shortly. Pasteur believed that the poison was brought to the surface by the upturning of the earth by earthworms.

**Pathology and Morbid Anatomy.**—Contrary to what one would expect, *rigor mortis* is prominent. The post-mortem changes may be summed up in a general way as consisting of infiltration of the connective tissues, and hæmorrhagic extravasations in various portions of the body; the special seat of lesions varying according to the portions of the body bearing the brunt of the disease. Thus, when the original sore is about the head or neck, the lymphatics of the neck are infiltrated, and their section shows them to contain hæmorrhagic extravasations. When the intestines are involved, there is a serous or sero-hæmorrhagic extravasation into the peritoneal cavity, and the surrounding connective tissue is jelly-like and of a yellowish-red color.

The blood itself is of a dark color. The mucous membranes are often injected.

In the intestines themselves may be found solitary or numerous infiltrations ranging in size from that of a lentil to that of a pea or coffee-bean, and presenting a grayish or greenish-yellow surface with a necrotic centre.

The spleen is always enlarged, often indeed to four times its normal size. Its distension with blood often reaches such a degree as to lead to its rupture. The specific bacteria are always found more abundant in the blood of this organ than elsewhere.

The anthrax bacillus may be found generally distributed throughout the blood. It consists of motionless rod-like bodies ranging in length from 5 to 12  $\mu$  in length, and 1  $\mu$  in breadth. They are somewhat larger at their extremities than at their middle, at which latter point they present the appearance of a transverse segmentation. These bacilli are readily destroyed by heat and dessication. In the cultivation of the bacillus endogenous spores are formed, which ultimately are freed and reproduce the rods. Spores which do not form in the living tissues, are very resistant, as they are unaffected by a short exposure to as high a temperature as 212° F., and prolonged immersion in a 5 per cent. solution of carbolic acid.

**Symptoms.**—The great rarity of anthrax in man in enlightened countries would seem to justify but a short description of its nature and phenomena in a practical text-book on medicine. But inasmuch as no one knows at what time it may be his unpleasant duty to attend one of these cases, and especially as early recognition of the disease and prompt and efficient treatment greatly reduce the mortality, a full description of the phenomena of anthrax in its various phases seems necessary.

In studying the symptomatology of this disease, it is well to divide the cases into two classes, according as the infection takes place on the external surface or through one of the viscera, notably the lungs and intestinal tract. In the former instance, single cases are observed, inoculations affecting only the individual. In the latter, the disease appears epidemically or in groups of cases, and arises from the ingestion of meat taken from affected animals, or the inhalation of dust from infected wool, hair, hides, etc. Taking first *external anthrax*, we find it making its first appearance on some portion of the body not covered by the clothing. It manifests itself by a small pimple with itching and burning, and this often within a very few hours after inoculation. Under no circumstances does the incubative period last longer than four days. This pimple or papule rapidly enlarges, and is surmounted by a small vesicle which soon bursts, the resulting raw surface becoming necrotic. The surrounding connective tissue becomes infiltrated. The surface over this infiltration is indurated and of a dark or bluish red. The periphery of the lesion is marked at times by a yellowish ring on which are set numerous minute vesicles. The slough which started at the central vesicle extends to the entire mass, finally separating, leaving an open ulcer, which, if a favorable result is to ensue, heals by granulation.

During the first twenty-four or forty-eight hours after the first appearance of the anthrax only the above-described local phenomena are present. The patient may be able to go about and to attend to his usual occupation. Constitutional symptoms now set in, and are of the character observed in toxæmia generally. Their course is rapidly progressive, so that death may result within eight or ten hours after their appearance. In other cases they are more prolonged, finally ending in recovery. Metastatic lesions are frequently met with, and the lymphatic glands adjacent to the primary lesion are inflamed and enlarged.

*Internal anthrax*, as already stated, may involve either the intestines or lungs, then receiving the names respectively of *intestinal mycosis* and *wool-sorters' disease*. The clinical phenomena of both conditions were recognized long before their pathological nature was known. The symptoms of intestinal mycosis are those of an acute poisoning. They appear within eight hours after eating the diseased flesh, and consist of chill, prostration, headache, pain in the abdomen, nausea, vomiting, and death within a very few days. No cutaneous manifestations of anthrax appear unless life is prolonged for eight or ten days.

WOOL-SORTERS' DISEASE is characterized by the symptoms of ordinary bronchitis with difficult respiration and profound nervous depression. Pains in the back and legs are prominent. In many cases the constitutional symptoms are absent until within a few hours of death, the patient being about and attending to his usual occupation until that time. The general features of the case may, however, be described as a rapidly fatal pneumonia with heart-failure.

**Diagnosis.**—The recognition of external anthrax is a comparatively easy matter, the principal diagnostic difficulty being its differentiation from ordinary carbuncle. The latter condition occurs more frequently on the back of the neck and on covered portions of the body. It pursues a decidedly more indolent course, and is far more painful than anthrax. Carbuncle proceeds from a number of points, its openings having been compared to the lid of a pepper-box. In anthrax the lesion spreads from one parent nucleus so to speak, reaches its acme within a very short time, and is followed by constitutional symptoms and metastatic lesions. The occupation of the patient is an important diagnostic guide in every case.

Intestinal mycosis cannot be recognized by the symptoms alone. Attending circumstances may here lead to the formation of an opinion. In doubtful cases microscopical examination of the blood for the bacillus anthracis, or inoculation experiments on lower animals, may give a decided result.

Wool-sorters' disease also depends for its positive recognition upon the discovery of anthrax bacilli and positive results from inoculation, together with a knowledge of the exposure of the patient to the causes of the disease. It has been said that wool and hair imported from South America are especially dangerous in the propagation of anthrax.

**Prognosis.**—Untreated anthrax of any variety is an exceedingly dangerous affection; over one-half of the cases of the external variety die. Recovery from either intestinal mycosis or wool-sorters' disease is the exception rather than the rule. Careful observations of external anthrax prove that the disease in the beginning is but local, and that the really dangerous toxæmia is but secondary, and is preventable. Proper treatment of this variety greatly reduces its mortality, as low a death-rate as 5 per cent. having been reported.

**Treatment.**—Prevention is, of course, always the most desirable. This can only be secured through proper disposal of the bodies of diseased animals. The recommendation that they be given deep burial is not sufficient, for one never knows when the progress of business enterprises may lead to disturbances of the soil to even a greater depth than any careful burial demands. The spores of the disease resist the most powerful germicidal agents unless carefully applied. In the ground they retain their virulence for years. Complete cremation of the bodies of all



animals dead of anthrax should be practised. Under no circumstances should economy leading to the preservation of their hides, hair, bones, etc., for industrial purposes, be countenanced.

As soon as the nature of the external lesion is recognized, it should be thoroughly excised, and the raw surface thus exposed, just as thoroughly cauterized by the galvano-cautery, pure carbolic acid, nitric acid, or caustic potash. Applications of irritants like iodine or nitrate of silver are absolutely useless. Recommendations involving complicated incisions and cross-incisions have been given; but the principle involved is early and complete destruction of the initial lesion which acts as a focus for the dissemination of the poison.

Internally, *lachesis*, *crotalus*, *anthracinum*, *arsenicum*, and *secale*, may be thought of.

Pasteur has proposed that animals liable to exposure to the poison receive protective inoculations. Acting on this line he has devised a means of mitigating the action of the poison, and protective inoculations with his preparation have been made. By this means animals have lost their susceptibility to artificial cultures of anthrax bacilli, but Koch has shown that no protection is obtained from the disease as ordinarily received.

## RABIES.

**Synonyms.**—Hydrophobia; lyssa; la rage; Hundswuth. The word hydrophobia, so commonly used to designate this affection, is not advisable, as rabies may exist without the presence of hydrophobic symptoms. Fear of the effort to take water, and even of the sound or sight of water, *i. e.*, hydrophobia, may be present in various diseases. In the rabies of dogs, it is not at all a constant symptom.

**Definition.**—An acute, infectious disease of the lower animals, affecting especially those of the canine species, transmissible to man by inoculation, and dependent upon an unknown, non-volatile poison. It is characterized, clinically, by a long period of incubation, and paroxysmal seizures dependent upon increased irritability of the medulla, ending in a paralytic stage and death.

**History.**—A disease presenting the horrible features of hydrophobia terminating fatally so invariably, cannot have failed to have attracted attention from the beginning. So we find mention of it in very early literature. Aristotle, writing 322 B. C., gives graphic accounts of it as it affects dogs, but states that men are not subject to it. The old Greek authors likewise make mention of this disease. It seems, however, to have escaped the watchful eye of Hippocrates. Galen knew of its occurrence in the human being, and remarked on its fatality. The disease seems always to have been surrounded by considerable superstition, which has lasted to the present day.

**Pathology and Morbid Anatomy.**—There is nothing specific in the lesions found. The most important changes have been discovered in the bloodvessels, especially those of the cerebro-spinal system. Hyperæmia, especially in the ganglia at the base of the brain, minute aneurismal sacculations of the vessels, and here and there punctated hæmorrhages, are the most important. The veins are distended, and there may be thrombosis and perivascular infiltration with leucocytes. The mucous membranes of the pharynx and stomach are hyperæmic, in fact, the same may be said of the entire alimentary as well as of the respiratory tract. The hyperæmia may pass on to the development of points of hæmorrhage in the nervous tissues. Most of the organs of the body are more or less engorged with blood, and their tissues manifest parenchymatous degeneration. Pasteur has found the virus freely in the nervous tissues, but not in the viscera.

**Etiology.**—There is but one cause for hydrophobia, and that is inoculation with the virus from an animal already subject to the disease.

It cannot, by any possibility, arise spontaneously, either in man or in the lower animals. In practically all cases, the inoculation is by the bite of a rabid animal. Exceptionally only does the virus gain entrance by other accidents. It cannot occur as the result of the bite of a non-rabid dog, angry though the animal be at the time of inflicting the injury. The possibility of communication by coitus has been argued without positive result.

Inoculation is the only means of transmission. Animals fed on different portions of the bodies of animals dead with hydrophobia, have failed to contract the disease. Dogs have been placed in stalls on straw used by rabid animals, and have failed of infection.

The disease may be transmitted from dog to dog, from dog to man or any of the lower animals, even those of herbivorous habits; or from man to man or the lower animals.

Hydrophobia, as it occurs in man, is the result of the bite of a dog, in 90 per cent. of all cases; in 4 per cent. it is caused by the bites of cats; in 4 per cent. by wolves, and in 2 per cent. by foxes. Not every one bitten even by a rabid animal contracts the disease. The possibility of infection depends very largely on the part of the body injured. Thus if the bite be inflicted on a covered portion of the body, in which case there is the opportunity of wiping the virus from the teeth before they puncture the flesh, a large proportion of those bitten escape a serious result. If the bite be upon the face, infection is pretty sure to occur. Taking the figures presented by Bollinger, 90 per cent. of bites on the face, and from 20 to 30 per cent. of those on the extremities, result in the development of the disease.

Among the superstitious causes assigned to rabies in dogs has been ungratified sexual desire; a cause which is readily shown to be without foundation in fact.

There are some medical men who look upon hydrophobia solely as an imaginary disease, the result of fright. This view is controverted by its occurrence in children and others who know nothing about it, and have not been anticipating it.

**Symptomatology.**—IN ANIMALS. Hydrophobia is remarkable for its long stage of incubation, during which it is believed to be inoculable. The most reliable investigations show that in four-fifths of the cases, the disease appears within two months after the bite; in 16 per cent. of the cases it comes on between two and three months. The longest period of incubation known in dogs is eight months. As occurring in dogs, we may follow the plan adopted by Bollinger, and divide the disease into two types: First, the violent or maniacal form, and second, the sullen form. The first of these is characterized by three stages, as follows: (1) prodromic or melancholy stage; (2) irritative or maniacal stage, and (3) paralytic stage.



(1) **PRODROMIC STAGE.** This presents but little that is distinctive. It may last all the way from a few hours to two or three days. During its continuance, the animal is noticed to exhibit marked changes in disposition. It is more or less hyperæsthetic. Its sexual instinct seems to be stimulated. It is weak and tremulous in the hind portion of the body. Then comes the violent stage.

(2) **VIOLENT OR MANIACAL STAGE.** In this the symptoms are of a spasmodic character. The animal presents still further changes in its manners. It refuses all food, and exhibits a very strong disposition to bite at anyone and anything coming in its way. It breaks away from its home, runs about, often inflicting havoc in its path. It is believed to be delirious, and affected by hallucinations. Evidences of the latter are said to be found in its biting and snapping at imaginary objects in the air. Insensibility to external impressions seems to exist at times. At no time in the course of the disease in dogs is there the slightest dread of water; indeed the animal is often thirsty, and seeks to allay that thirst at any opportunity; nor is there the slightest sensibility to light, sounds or drafts of air. This stage lasts from three to four days.

(3) **PARALYTIC STAGE.** The paroxysms become weaker, and the animal loses control over the hind legs. It emaciates rapidly. It may still persist in biting at surrounding objects as much as its weakened state will permit.

In the sullen type of the disease, the maniacal stage is absent, the melancholic first stage merging directly into the paralytic. These cases run a much shorter course than those of the maniacal type.

**IN MAN.** As in the lower animals, the stage of incubation is remarkable for its variations in different cases. Again using Bollinger's figures, in 6 per cent. of the cases, it is from three to eighteen days; in 60 per cent. from eighteen to sixty-four days; and in 34 per cent. it exceeds sixty days. In rare cases it is said to have been prolonged to two or more years. Such cases should be viewed with great suspicion, as they are not based on reliable information. This stage is free from symptoms as a rule.

These great variations in incubation have been discussed. Horsley states that the incubation is shorter in children than in adults; when the part bitten is an uncovered portion of the body; and when the wound is a punctured one, or there are extensive lacerations. The animal conveying the disease exerts some influence, wolves being the most dangerous.

(1) **PREMONITORY STAGE.** This is characterized by evidences of constitutional disturbances, these partaking, however, of a very indefinite nature. The wound, which had healed completely, sometimes becomes red and gives evidence of irritation. The patient is ill-natured, restless and apprehensive. He is sensitive to light and air. Sometimes the

inability to drink is present at this time; indeed all other symptoms may be absent, and this be the first manifestation of the disease.

(2) **STAGE OF EXCITEMENT.** In this we find the active symptoms of the disease. In a general way we may group its symptoms under maniacal excitement, restlessness and hyperæsthesia. It is usually the latter that attracts attention. Water especially provokes paroxysms of spasm of the muscles of the pharynx and respiration; even the sight of it, or the mere mention of the word, is sufficient to occasion the greatest distress. A mere breath of air, a slight rustling sound, or a mere touch, is sufficient to provoke reflex spasms.

The paroxysms of spasm last from one-half to three-quarters of an hour. During their continuance the mind may be clear; but usually there is more or less delirium, and many hallucinations. The patient may see imaginary objects, or believe that his present troubles are the result of the machinations of his attendants, whom he accordingly tries to injure. In the intervals between the attacks the mind is usually perfectly clear, the patient being fully cognizant of his serious condition, and of his attempts to injure his friends.

Oppression of breathing and even severe dyspnœa are present.

The saliva is increased in quantity; and owing to the difficulty in deglutition, it is not swallowed, but escapes by the mouth.

(3) **PARALYTIC STAGE.** In this the patient becomes quiet, and the spasms subside. He may now be able to allay his thirst, which he does with avidity. Gradually general paralysis appears and the patient dies.

**Diagnosis.**—This is made by a knowledge of the entire clinical history of the case. Especially by the manifestations of hyperæsthesia referred to the muscles of deglutition and respiration. Cases of hysteria simulating hydrophobia very closely do occur, and may prove very difficult of recognition. There will always be found in such, certain features which the experienced eye will recognize as indicative of the less serious disorder.

**The Prognosis** of hydrophobia is invariably unfavorable. This statement applies to both man and the lower animals. Cases of recovery are instances of incorrect diagnosis.

**Treatment.**—The most important point is prophylaxis. This can only be secured with any degree of certainty by muzzling or exterminating all dogs.

Immediately on the reception of a bite, the wound should be excised or thoroughly cauterized with fuming nitric acid. If facilities for this are not at once available, the wound should be sucked. If so situated as to be inaccessible to the patient himself, it may be done by a third party without danger, unless there be present open wounds of the mucous membrane of the mouth. Cupping may be resorted to for the purpose of withdrawing blood from the wound.

PREVENTIVE INOCULATIONS. These originated with Pasteur. This eminent authority depended for his virus upon the fact that the central nervous systems, especially the spinal cords of rabid animals, contained the virus in its most concentrated form. Making inoculations on series of rabbits, he found that the virus became progressively more active as the inoculations increased, until at the end of twenty-five generations, virus which originally would have taken twenty days to produce the disease, now did so at the end of seven days. When the spinal cords of these animals are preserved in dry air the virus gradually loses its intensity. In practising protective inoculations, cords preserved for twelve or fifteen days were first used. Then on succeeding days, cords preserved for a less and less length of time, until finally the most active ones were used. Animals treated in this way lost their susceptibility to virus that was almost inevitably fatal to unprotected animals. This method of treatment has been followed out in a large number of cases for the prevention of hydrophobia among those bitten by rabid animals, and with apparent success, the mortality under the treatment being less than 1 per cent. of those applying for and taking it.

In the developed disease there is little to be done but to diminish the patient's sufferings as much as possible. This may be done by the free use of chloroform or morphia.

*Cantharis* has been recommended by both schools of medicine and is undoubtedly homœopathic to the disease. It seems to be as inefficient as any other remedy.

*Belladonna* presents in its symptomatology a close resemblance to rabies, and may be administered.



## GLANDERS.

**Definition.**—An infectious disease of the horse which is occasionally communicated to other animals, including man, and which is characterized by the development of nodular growths in the nose (glanders) and in the subcutaneous lymph structures (farcy).

**History.**—A knowledge of this disease as affecting the horse was had from great antiquity, notwithstanding which its communicability to man was not recognized until 1783, when Osiander reported a case of glanders in the human being. In 1812, Lorin, a French surgeon, reported a case of farcy. The patient, remarkable to state, recovered after an illness of a little over two weeks. Since that time numerous other cases in man have been reported, until now the infection excites but little comment. Rayer, in 1837, published a monograph on glanders, he being the first to fully describe the disease as it appears in man. The description he gave fifty years ago was so complete that it can be added to but little at this day. The pathology of the disease was investigated by Virchow in 1858 and 1863. Some of the older ideas concerning the pathology of the disease are, to say the least, amusing in the light of modern scientific research. Thus it has been at different times assigned as the cause of syphilis, tuberculosis, diphtheria, pyæmia and other infectious diseases; eminent authorities giving their support to these fallacies.

In 1882 Lœffler and Schütz isolated and cultivated a bacillus with which they were able to produce the disease by inoculation. This bacillus grows readily upon quite a variety of culture media. It is a rod-like body, 2 to 3  $\mu$  long and 0.3 to 0.4  $\mu$  broad, and very often carries a spore on the end. It may be found in the discharges from the ulceration, in the nodules, and in the blood.

**Etiology.**—The etiology of glanders is included in one word, *i. e.*, infection. Cases have been said to have arisen spontaneously, but such statements are doubtless due to inaccurate observation. Man usually contracts the disease from infected horses. Those having much to do with these animals in their daily pursuits are, of course, the class furnishing nearly all the cases. So true is this statement, that glanders in man may almost be regarded as an industrial disease. The variety of ways in which the poison may be taken into the body is almost endless. Generally, infection takes place by the discharges from the infected animal finding lodgment on a cuticular abrasion. Some claim, however, that a wound of the integument is not a necessity to inoculation, and that the discharge coming in contact with the healthy skin may be

absorbed through the hair follicles, and thereby infect the unfortunate subject.

There can be no question concerning the danger of the discharge from animals coming in contact with mucous membranes of man. In one case which came under my notice, the discharge thrown off by a snorting horse was carried backward into the face of the driver by a sharp wind. Mackenzie, Osler and others have reported cases originating in a similar manner. When one considers the commonness of this habit of horses and the fair degree of frequency with which glanders in these animals escapes observation, it is rather wonderful that this disease in man is one of the rarest, and not one of the most common.

The susceptibility of man to infection is not regarded by Bollinger as very great, he contending that individual susceptibility to infection is necessary. Many persons repeatedly exposed fail to take the disease. This may explain the experiments of Decroix, which by some writers have been discredited. This observer, with what must be regarded as undue zeal, ate freely of the flesh of a glandered horse, taking it raw and cooked in various ways, without becoming infected. Semmer experimenting in the same direction, fed animals in a menagerie on glandered meat without result. Gerlach, however, reports a case in which a cat about an anatomical museum ate the flesh of a horse dying of glanders, and was itself affected.

**Pathology and Morbid Anatomy.**—The special anatomical feature of glanders is a granulomatous tumor, which appearing in the nose, tends quickly to ulceration, and occurring beneath the skin, to the formation of abscesses. When infection becomes general, various pathological changes are found in the viscera. These are in general those that may invade the entire body, involving as they do not only the skin and mucous membranes, but the muscles, viscera, periosteum, etc. In each of these parts they pursue identical courses, enlarging, softening, and suppurating.

**Clinical Course.**—The incubation period varies from three to five days. Much longer periods have, however, been reported. If the disease has resulted from infection through a cutaneous abrasion, local symptoms consisting of pain, redness and swelling, are manifested. The surrounding lymphatics take up the poison and show evidence of involvement. With these local symptoms there may appear constitutional symptoms, as fever, pain in the head and limbs, general malaise, etc. In some cases the latter may precede the local condition at the point of infection. Now the characteristic lesion of the disease makes its appearance. As already stated, it consists of nodular growths beneath the skin. These vary in size from that of a pea to that of a walnut. They may even increase to such an extent as to be dignified by the designation of tumor. They soften and break down into abscesses, which leave deep

torpid ulcerations. These latter may extend through all the superficial structures, exposing tendons, muscles, and even the bones.

The nasal cavity is very frequently affected. The mucous membrane is greatly swollen and studded with the characteristic nodules with their subsequent ulcerations. There is an exceedingly offensive purulent discharge. The swelling and redness extend to the accessory mucous membranes, which likewise ulcerate. Even the mucous membranes lining the alimentary and respiratory tracts may become affected, thus leading to additional important symptoms. Infection of the nose may develop as late as the third week of the disease. The salivary glands and the lymphatics in the cervical region may be involved, and the former especially may suppurate.

As the disease progresses, the patient emaciates and tends to a condition of intense asthenia. The temperature rises to 104° F. or more; the pulse grows weak; and delirium and finally stupor supervene. Urination and defecation become involuntary, and the patient dies from exhaustion. The average course of acute glanders as above outlined is sixteen and one-half days.

Chronic glanders presents but little variation from the foregoing other than intensity of symptoms and rapidity of progress. The prominent symptoms in such cases are the nasal ulcerations and the ozæna-like discharge, and the cutaneous abscesses and ulcers which display no tendency to repair. The chronic ailment starts as such; it is not a continuation of the acute disease. In fact, the general tendency of change is from the chronic to the acute form.

**Diagnosis.**—During the first few days of an acute attack, typhoid fever may be suspected. The appearance of the characteristic nodules then clears up the case. Bacteriological examination is always practicable. A cover-glass upon which a little discharge has been quickly dried may be stained in a solution of methylene-blue of the strength of one part to one hundred of distilled water. It is then washed in a solution of acetic acid (1: 3), allowed to remain in absolute alcohol for a few minutes, and finally mounted in Canada balsam. If the bacteriological examination is not satisfactory, inoculation experiments may be tried, guinea-pigs being the preferable animals for this purpose.

Cases will arise in which a diagnosis by the symptoms alone is impossible. In such, inoculation experiments are necessary before a conclusive opinion can be offered. Semmer and Waldinirow believe that the surest means consist in the inoculation of some of the morbid products into cats or guinea-pigs, and the making of control experiments with pure cultures on potato. Veterinary surgeons have of late relied, on the recommendation of Helman, on the hypodermic administration of mallein. This is a substance obtained by extracting cultures of the glanders bacillus with glycerin. Before using it is diluted by adding



nine times its volume of one-half per cent. of carbolic acid solution. Injected into horses suffering from glanders, it produces rise in temperature of from one to six degrees, and forms at the seat of inoculation a tumor which increases rapidly for two or three days and then disappears. This diagnostic measure has now received official approval in the German army.

Syphilis may be confounded with the chronic form of the disorder. We are enabled to differentiate by the discovery of the bacilli of glanders in the one instance, and the curative effect of iodide of potassium in the other.

**Prognosis.**—This is always most unfavorable. Some have even denied the possibility of a cure in the acute cases. When, however, the disease is limited to the cutaneous surface, there may be a possibility of recovery by prompt use of surgical measures. Chronic cases recover in about one-half the cases. Even then the patient's health may remain undermined for the balance of his life.

**Treatment.**—Prophylaxis is of the utmost importance. If seen early, the infected spot should be excised or destroyed with suitable caustics. If the discharges have been lodged on a mucous membrane, Ivins recommends the thorough irrigation of the same with a permanganate of potassium solution. All infected animals should be killed without delay. All discharges from those infected must be destroyed at once, for it must be remembered that the disease may be conveyed from man to man. The nose and all affected parts should be repeatedly cleansed with antiseptic solutions. All accessible lesions should be treated according to indications on sound surgical principles. The patient's strength must be supported by a carefully selected dietary, and perhaps by stimulants.

Serum therapy seems to be of some value in glanders, and inasmuch as other measures thus far recommended are not such as to offer encouraging results, is well worthy of trial. "Hell obtained from horses, which had recently passed through an attack of glanders, blood-serum which he sterilized and injected in doses of forty, rarely eighty grammes (one and one-quarter to two and one-half ounces), horses receiving in three weeks 320 grammes. In one regiment fifty-four horses were so treated. In one squadron twelve horses were injected, and after this no more cases of pulmonary glanders appeared. In cases of horses already affected, the disease was brought to a standstill in three days. He adds that repeated injections are necessary for protection, and for cure in early cases, the serum must not be used too sparingly. Hell's opinion, as well as Toepper's, is that the serum of horses which have had glanders has both protective and curative properties against the disease. For the latter purpose, 100 grammes (three and one-quarter ounces), repeated two or three times, are required."

Experience with remedies in the treatment of glanders seems to be

decidedly limited. Recommendations made by Hering and Hughes are based entirely upon theoretical considerations. The former recommends *kali bichromicum*, *mercurius* and *crotalus*; the latter, *arsenicum*, *phosphoric acid*, *calcareo* and *sulphur*. Ehrhardt reports one case cured with *arsenicum*. Dr. E. C. Price has used *graphites* in several cases in horses with considerable success. Jousset recommends *aconite* and *arsenicum*. *Kali bichromicum*, as Hughes truly remarks, "is exquisitely homœopathic to the respiratory—especially to the nasal—affections of the disease, and hardly less so to its cutaneous phenomena, as may be seen on reading the 'skin' section of Dr. Drysdale's arrangement of the drug in the *Hahnemann Materia Medica*." Hughes quotes Moore, an English veterinary surgeon, as authority for the successful application of the remedy in practice.

Ivins, quoting from literature, says that three cases have been cured by mercurial inunction and massive doses of iodide of potassium. Owing to the occasional close resemblance of glanders to certain phases of syphilis, one may question the diagnosis of these cases. The same authority indicates as his preference for local treatment of glanders affecting the nose, the "washing of the ulcers with a 10 to 20 per cent. carbolic acid solution, red permanganate of potassium solution or fifteen-volume peroxide of hydrogen."

## MOUTH AND FOOT DISEASE.

**Nomenclature.**—Aphthæ; epizoöticæ.

**Definition.**—An acute but mild infectious disease of certain of the lower animals, communicable to man, and characterized by the formation of vesicles and ulcers on the mucous membrane of the mouth and about the feet.

This disease presents quite a remarkable feature. Its commonness in the lower animals is attested by the fact that in one epidemic in England alone no less than 700,000 animals suffered; during the same year it is believed that fully the same number were affected in France and Germany. It seems to be readily communicable to man, and yet but few physicians appear to have ever seen cases of it. The only possible explanation of this seems to be either that the susceptibility of man to the contagion is very slight, or the mildness of the disorder, taken in connection with the fact that most of those liable to infection belong to a class proverbially careless in matters pertaining to personal health, prevents the subjects of it from consulting physicians.

**History.**—As an epidemic disease of animals, mouth and foot disease has been known for centuries, and was formerly confounded with anthrax and actinomycosis. Its communicability to man was not attested until 1764, when Sagar described a case originating from drinking the milk of an infected cow. The contagiousness of the disease was announced by Hertwig in 1834. This observer with two other physicians drank freely of the milk from infected cows, with the result of contracting the disease. Others have repeated the experiment with like result; while some have partaken of the infected milk with impunity.

**Etiology.**—Foot and mouth disease can occur only through contagion. The exact nature of the poison is as yet unknown. It originates in animals, notably in cattle, sheep and pigs, and less frequently in horses and dogs. Whether communicable through the air or not, is undecided. The poison is very tenacious of life, having once infested a stable, it persists therein for years.

It is communicated to man most frequently by the ingestion of milk from diseased cattle. It may also arise from the handling of articles of clothing, farm implements, etc. Direct infection may take place from milking diseased cows.

**Symptomatology.**—(a) IN ANIMALS. The first manifestation of foot and mouth disease in the lower animals is fever, which is associated with mental dulness, and is shortly succeeded by catarrhal inflammation of



the mucous membrane of the mouth, with salivation. There next appear upon the mucous membrane of the mouth whitish-yellow vesicles, at first transparent, later becoming turbid. These vesicles rupture, forming small ulcers, which may coalesce. At the end of from three to six days the lesions will have completely healed. Along with the sore mouth, there develop similar lesions about the hoofs. The contiguous parts become red and sensitive; vesicles form; these burst and produce ulcers, on which crusts are formed. Healing takes place beneath these scabs and is completed by the fourteenth day after the inception of the disorder. The milk presents certain important changes. It is always greatly diminished in quantity and shows a yellow colostrum-like appearance.

(b) IN MAN. After an incubation ranging from three to five days fever sets in, associated with slight headache and dryness of the mouth. Then the vesicles appear on the mucous membrane, and follow the same course and with the same concomitants as in the case of the lower animals. Pain is probably greater, and the tongue is not infrequently very much swollen.

The digestive function is next disordered, and a vesicular eruption then appears upon the fingers and hands. It frequently affects the parts about the finger nails, but never destroys these appendages.

Sometimes the vesicles involve the fauces. In others, the eruption is general. In the mildest cases the symptoms are merely those of a catarrhal stomatitis.

**Diagnosis.**—The diagnosis is established by the knowledge of the prevalence of mouth and foot disease among animals, and the fact that large numbers of persons are simultaneously affected, and have partaken of the milk of the diseased cows.

**Prognosis.**—This is nearly always favorable, both for man and beast. The only exceptions to the ordinarily mild course of the disorder are found in young children previously debilitated, who may occasionally succumb. It has been suggested that these unfortunate results are as much due to chemical changes in the diseased milk as to direct infection.

**Treatment.**—So far as infection from milk is concerned, the disease may be prevented by boiling that liquid, which effectually destroys all the contagious elements.

Other treatment should be purely symptomatic. The mouth may be cleansed regularly with a solution of borax. Internally, *rhus*, *mercurius* and *croton tiglium* may be thought of.

## ACTINOMYCOSIS.

**Definition.**—A specific affection of a chronic inflammatory character common to man and cattle, and caused by the actinomyces or ray-fungus.

**History.**—This peculiar affection, only recently understood, is due to the entrance into the body of the vegetable parasite known as the actinomyces or ray-fungus.\* In 1877 Israel, of Berlin, published the first distinct description of the disease as it appears in man, although cases must have been observed in the past, but were probably generally classed as tubercular developments. In 1878 Ponfick showed that a similar affection appearing in cattle was due to the same cause. Since that time a number of observers have reported carefully studied cases. Attempts have likewise been successfully made to cultivate the fungus by Israel, Bostrom and Wolff, and the latter has succeeded in inoculating the disease, using for this purpose pure cultures.

This fungus is visible to the unaided eye. It is a spherical, yellowish, granular mass, said to average about one-fortieth of an inch in diameter, but which may be even one-eighth or one-tenth of an inch. The more recent granules are soft and of a grayish color; the older ones are firmer in consistency and present the characteristic yellowish color. Some of the oldest developments may be of a yellowish-brown or greenish tint. An agglomeration of these granules may develop masses as large as an orange.

Local lesions of an inflammatory character are excited by the development of this fungus in the tissues, or rather, as is supposed, as the result of a combined infection, *i. e.*, the penetration of pyogenic organisms also.

Examinations of these granular masses with the microscope demonstrate them to be composed centrally of a closely woven mass of mycelial threads. Arranged radially to this core are threads which often divide dichotomously, but all of which terminate in clubbed extremities. Variations from this typical arrangement are not uncommon. The early irritant action of the fungus leads to tissue hyperplasia, a little cell-knot consisting of round epithelial and even giant cells resulting. These granules bear a close resemblance to miliary tubercles. From these centres of irritation the cellular tissue is stimulated to active development and a firm growth results, which may, for a time, be easily mis-

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\* In his studies of the affection of cattle known as "big-jaw," etc., Bollinger in 1877 discovered the presence of a peculiar fungus, which Hartz, the botanist of Munich, to whom specimens were presented, designated as the actinomyces or ray-fungus.

taken for a malignant tumor. Suppuration, softening and discharges, constitute the final stage of the process. Bone as well as the soft tissues may be attacked.

The mode of infection of the body is still uncertain, as the fungus has not yet been discovered external to the body. It seems probable that it is taken in with food, and finds lodgment in abrasions of the mouth or pharynx, and by aspiration or swallowing is carried to points lower down. Israel found fragments in the crypts of the tonsils and in carious teeth. Some growths have seemed to originate from the teeth.

The fungus is supposed to be taken into the mouth in vegetable food, especially the cereals. Numerous observations by several authorities support this statement.

From the location of primary development, the parasite may be disseminated, developing foci in various organs, but most frequently in the subcutaneous and intermuscular connective tissue.

**Clinical Varieties.**—The disease has been described as it occurs in connection with the mouth, the respiratory apparatus, and the alimentary canal. When accessible to examination, a tumor is found, of slow growth, attended by little pain, and which ultimately suppurates. The resulting sinuses discharge a sero-purulent fluid, possessing little odor, and containing the characteristic granules. With the growth of the mass, if upon the lower jaw, the bone becomes enlarged, the neck changes in shape, and the cutaneous and subcutaneous connective tissues transformed into a thick, cicatricial layer. The fatal result is due to asthenia consequent upon protracted suppuration, unless occlusion of the trachea, œsophagus, or large vessels, occurs earlier in the course of the disease.

In pulmonary actinomycosis, the irritant may develop a form of bronchitis which resembles putrid bronchitis. The sputum separates into two layers, not into three, as in the putrid variety. The upper stratum is clear, the lower is turbid, and contains the ray-fungus. When the lung parenchyma is involved, the course may be either rapid or slow. If the former, the symptoms simulate those of pneumonic fever; if the latter, tuberculosis.

The intestinal manifestations consist of grayish patches, which contain granules of a yellowish or brownish color. Small ulcerations have been observed in the intestinal walls. Here and there may be swellings, some of which have suppurated. Perforation and discharge into the peritoneal sac have been observed; also adhesions and discharge through the abdominal parietes. Involvement of the liver has been reported as primary development, but it is usually secondary to the intestinal form.

**Diagnosis.**—The resemblances between actinomycosis and pyæmia are many and pronounced, but the presence of the fungus in the pus of the former enables us to differentiate. Indeed, in all forms of the affections we must rely upon the specific fungus for a diagnosis. It must



not be forgotten that primary actinomycosis of the brain has been observed by Bollinger, and that Sharkey and Ashland reported primary involvement of the liver. The presence of vegetable fibres in any purulent discharge is considered by Fisch as suggestive of the nature of the disease.

**Prognosis.**—When the disease involves the head, neck or structures readily accessible to surgical measures, a favorable result is not infrequently secured. When actinomycosis involves the thorax or the abdomen, the prognosis is very unfavorable, though not necessarily fatal. Involvement of the anterior abdominal wall offers a better prospect than does that of the retroperitoneal structures. When functionally unimportant organs are involved, the disease may last for many years. Pyæmic cases are very unfavorable.

**Treatment.**—**PROPHYLACTIC.** It is advised to destroy the tissues of all slaughtered animals suffering from this disease. Food calculated to injure the mouths of cattle should be avoided. Proper care of the mouth and teeth must be regarded as preventive.

Thus far surgical treatment only has proven of use if we are to rely only on standard authority. Excision of the lesion or its destruction with caustics, especially nitrate of silver, has been most practised, and has cured a few cases. Abscesses must be opened and thorough drainage effected. Billroth claims to have cured one case with tuberculin. I know of no published homœopathic experience in the treatment of this disease; within one or two years, iodide of potassium has been strongly advocated as a valuable remedy in actinomycosis. Some even go to the extent of saying that it is specific. It is generally given in doses of fifteen grains daily. Some believe that the best results will be obtained when it is pushed to the extent of producing iodism.

## MILK SICKNESS.

Concerning milk sickness the most diverse views prevail, the very existence of the affection even having been called in question. It is a disease communicated to man from cattle, either by partaking of the flesh or of the milk or milk products of diseased animals. Milk sickness was at one time comparatively common in western countries; but with the advance of civilization and the clearing up of old wooded districts it has disappeared, and now is limited in the United States almost exclusively to certain regions of North Carolina.

Cattle are infected by grazing in certain well-wooded districts. It has been claimed by some that the special poison giving rise to the trouble is a baneful plant or mineral peculiar to the grazing ground. This supposition has been thoroughly disproved, and instead the theory that it is dependent upon a specific organism of unknown character has been advanced with very good reasons in support of it. Cases of milk sickness are observed generally in the late summer or early autumn, especially when the summer has been one with but little rain. Cheese and butter made from the infected milk are fully as dangerous as the milk itself.

Cattle affected with milk sickness are said to have the "trembles," or "slows," the former name having been applied because of the peculiar twitching or trembling of the voluntary muscles. The animal at first shows its illness by a listless or apathetic state. It refuses all food, although it partakes of water freely and with avidity. Its bowels are obstinately constipated. Exercise intensifies its symptoms, bringing on the characteristic tremor already referred to. If the enforced movements are continued, death may result. Other symptoms observed are those of profound prostration, the animal lying at full length on the ground, with cool skin and glassy eyes. Finally death results.

In man the same apathy and indifference are early shown. The most profound weakness is the initial symptom, and this enforces the patient to remain in his room. With this precaution, other symptoms may not develop for several days. Thirst is a prominent symptom. Persistent nausea, however, occasions the most inconvenience. The breath has a peculiar heavy odor, which by many is regarded as characteristic of the disease. The abdomen is flat and flaccid. The temperature is normal or subnormal, notwithstanding which the patient complains of feeling hot. In fatal cases the patient gradually sinks into a comatose state.

The treatment employed by old-school physicians who have had the most experience with the disease is eliminative in character, *i. e.*, mercurial purgation. I know of no published homœopathic experience.

## TUBERCULOSIS.

**Definition.**—Tuberculosis is an infectious disease attacking man and many of the lower animals, pursuing an acute or chronic course, and characterized by a variety of nodular and diffuse lesions, not limited to any tissue or organ, but which may be localized or widespread in their distribution. This newly developed tuberculous tissue undergoes a variety of secondary changes, the most constant being known as caseation or cheesy softening. This is often followed by ulceration, and less frequently by sclerosis or calcification. The *bacillus tuberculosis* is constantly associated with tuberculous lesions, and is without doubt the cause of the disease.

**THE BACILLUS TUBERCULOSIS.** We owe our knowledge of the bacillus tuberculosis to Koch, who first described it in 1882. His researches were so thorough that but little has been added by other observers to the description this eminent bacteriologist presented us in his earlier papers, notwithstanding an enormous amount of experimentation and anatomical investigation has been provoked by his discoveries. The constant association of this bacillus with tuberculous processes, our ability to separate and cultivate it external to the human body, and the development of tuberculosis in the bodies of animals into which it is introduced, may be considered as established facts, and sufficient to justify our considering this organism the cause of the disease. Every physician should read the original communications of Koch relating to this subject, as they furnish a marvellous demonstration of the results which follow strict adherence to scientific methods.

The tubercle bacillus appears as a minute rod with rounded extremities. Both its length and breadth are variable, especially the former, which is from 1.5 to 3.5  $\mu$ . The breadth is 0.2  $\mu$ . When stained the bacillus presents a beaded appearance, which has been attributed to the presence of spores, but which Sternberg states is the result of irregular staining. This irregular coloration leads to an appearance not unlike that of a chain of micrococci, especially if two or more of the bacilli are united. The bacillus is generally slightly curved, immobile, and stains slowly with the basic aniline dyes; but when once stained, retains its color with greater tenacity than other similarly constructed bacilli.

It is necessary that staining methods be employed in order that this organism may be made accessible to observation. Perhaps the most popular and satisfactory method is that devised by Ehrlich, who uses a fluid prepared according to the following formula: Add 5 cc. of pure



aniline oil to 100 cc. of distilled water; shake and filter to remove the excess of oil. To this aniline water add 11 cc. of a saturated solution of fuchsin and 10 cc. of alcohol. Sputum may be readily examined by placing a minute quantity of it between two cover-glasses and spreading it into a thin layer by pressure. The glasses are now to be separated by a sliding movement and dried. Then place the cover-glasses in the staining material for twenty-four hours, or, if more rapid results are desired, into a quantity of the stain which has been raised to the boiling point, the hot fluid coloring satisfactorily in a few minutes. The stain is next removed from all but the tubercle bacillus by washing for a few seconds in a mixture of nitric acid and distilled water (1-4), the acid is to be immediately removed by washing once or twice in distilled water. If it is desired to examine the specimens in Canada balsam, the cover-glass must be first placed in 95 per cent. alcohol for a few minutes. But if glycerin is used, this step is not necessary. If double staining is desired, instructions for the same will be found in any recent work treating of pathological histology.

**GROWTH OF THE TUBERCLE BACILLUS.** The tubercle bacillus, so far as we are at present informed, grows only in the body of man and a few of the warm-blooded animals. It develops most actively upon coagulated blood serum maintained at the normal temperature of the body. The growth is slow, requiring from fifteen to twenty days in which to develop appreciably upon artificial culture media. It forms thin, grayish-white, dry scales, without lustre, which may coalesce, forming an irregularly shaped pellicle. Dessication does not kill, at least not for long periods of time, but the bacillus is destroyed by exposure to boiling water for some minutes.

Koch has recently stated, that when the tubercle bacillus is fully exposed to the direct rays of the sun, it is destroyed in a period varying from a few minutes to a few hours, depending upon the thickness of the layer. It is also destroyed by diffuse daylight in about a week. The hygienic importance of this observation is very great.

Experiments conducted by Sawizky indicate that dry sputum preserves its infectious quality for about two and one-half months in ordinary dwelling-rooms.

J. Grancher and Ledoux-Lebard have made careful experiments upon the influence of temperature on the activity of the bacillus. The human bacillus, in distilled water, grew after being preserved at a temperature of 122° F. for fifteen minutes. A temperature in excess of 140° F. resulted in sterilization. A temperature of 212° F. for one-half minute was sufficient for sterilization. In a dry state the bacilli were still virulent after seven hours exposure to 150° F. and also after an exposure of three hours to 212° F. The bacillus of avian tuberculosis, in bouillon or distilled water, withstood a temperature of 122° F. for fifty minutes,

and 140° F. for ten minutes. A temperature of 158° F. to 212° F. destroys the germs in one minute. A temperature of 122° F. for fifteen minutes left the bacilli unchanged. Virulence was diminished after an exposure to a temperature of 140° F. for fifteen minutes, and ceased, as stated, at 158° F.

Sander states that the obstacles to the saprophytic cultivation of Koch's bacillus have been exaggerated. Growth, although slight, he has found successful upon various vegetables, especially upon acid potato.

Fischl has carefully studied the varieties of tubercle bacillus which have been described. He regards Koch's bacillus as a parasitic form of an originally saprophytic fungus, and avian tuberculosis as a subspecies, the result of the character of the soil upon which it grows. He further believes that the bacillus of miliary tuberculosis of murrain (*Perlsucht*), and that of avian tuberculosis may be differentiated. The avian bacillus loses its ability to produce tuberculosis when introduced into mammals, through the influence of the new soil upon its character. Virulent bacilli have been repeatedly found by Pizzini in the glands, especially the bronchial of persons who have manifested no evidences of tubercular disease. These bacilli had probably found their way from the respiratory mucous membrane, and were ready under favorable circumstances to develop tuberculous lesions. The same observation has been made by others upon children dead from diphtheria and various septic troubles, who had been free from any evidences of tuberculosis. These and other observations in the same line, suggest that in children the glandular structures within the chest are often the first attacked, and that this really constitutes the earliest evidence of the disease. H. Neumann mentions bronchial breathing in the upper dorsal region, and peculiar paroxysms of coughing, as most diagnostic; but considers a diagnosis as possible only in advanced cases.

The results of recent investigations are strongly suggestive that at least the pulmonary form of tuberculosis is not a simple infection. It appears probable that Koch's bacillus is the factor in the development of the typical lesion, *i. e.*, the tubercle; but that the associated destructive lesions of a bronchitic and pneumonic character are the result of the action of organisms possessing a greater power of destruction, and the ability to produce diseases of a more septic character than the bacillus tuberculosis. These associated organisms have been carefully studied by Petruschky, Cornet, and Kitasato. The result has been the finding of the streptococcus present in the majority of cases, and more especially in those attended by fever. In some cases the streptococci were also found in the blood and the tissues of many of the organs. If these observations are sustained we must look upon the hectic fever of advanced pulmonary phthisis as the same in nature as that associated with wounds, erysipelas, etc. Koch has graphically described the temperature curve

peculiar to this condition as the "streptococcus curve." These observations explain the clinical instructions of Koch which confine tuberculin to the treatment of the early stage of tuberculosis. Many clinicians have observed bad results from its employment during the later stages.

It has been suggested that the inflammatory changes attendant upon pulmonary tuberculosis may be the result of the action of the pneumococcus. In support of this idea we may quote the recent observations of Ortnier upon sixty-one cases, his deduction being favorable to this idea.

**History.**—The term, "tuberculosis," was first employed by Schönlein in 1839, to indicate a definite disease. The term, "tubercle," had long been employed to indicate nodular growths, but it was popularized by Bayle and Laennec, in the early portion of this century, as a term indicating the pathological lesion in tuberculosis. This application of the word was long influential in obscuring the true nature of the disease, too much importance being ascribed to the nodular feature of the lesion. The injurious influence of this nomenclature is apparent, even to this day. The word, "phthisis," is of Greek origin, and was suggested by one of the most prominent clinical features of the disease, *i.e.*, wasting.

A survey of the evolution of our knowledge of this interesting disease must prove of interest to every physician. The ancients had knowledge of a disease characterized by emaciation, cough, and suppuration of the lungs. Later, a great variety of nodular developments were called tubercles, but of the miliary tubercle they were ignorant; indeed, of tuberculosis, as we know it, they had no conception. From these early days until the beginning of the nineteenth century, there was some gain of knowledge in relation to the gross structural changes present in tuberculosis. In 1810 Bayle demonstrated the presence of minute granulations in many tissues (miliary tubercles), and suggested the possibility of their relation to phthisis. This was the first great advance, but it included the error of considering tuberculosis as a disease characterized by a specific deposit not dependent upon inflammation. This discovery was followed by persevering research upon the part of many observers to determine if definite anatomical peculiarities, which might be considered as special to tuberculosis, were present. The change which Virchow long after called caseation and demonstrated as present in lesions undoubtedly not tuberculous, was considered by Laennec (who was contemporary with Bayle) as the specific change, and he consequently looked upon all lesions presenting this degenerative change as tuberculous. Laennec recognized the isolated granules and the diffuse infiltration of tuberculosis, and also appreciated in some degree the relationship existing between pulmonary tuberculosis and processes which, at that time and even until the discoveries of Koch, had not been understood. The development of physical diagnosis by Laennec did much to aid in the diagnosis of pulmonary tuberculosis. It should be remembered that the



work of this great physician was not the completion of that begun and carried towards perfection by his predecessors, but that it was almost entirely original. Since 1847 the influence of Virchow's teachings has been greater than those of any other pathologist. He distinguished between tubercle and inflammatory and other lesions which had undergone caseation. He looked upon the miliary tubercle as the anatomical evidence of tuberculosis, and incited study into the minute structures of these granulations by Schüppel, Wagner, Langhaus, and others. But its origin and relationship to the infiltrated masses, as well as to certain general processes, viz., scrofula, were not to be conclusively determined by means of the microscope. The keynote to the proper method of investigation, which was to make clear this vexed question, which had been perhaps foremost in the minds of pathologists, was sounded by the discovery of Villemin in 1865, that tuberculosis could be communicated to animals by inoculation. While the doctrine of the infectious character of this disease has been antagonized, it has grown steadily in favor, surmounting all opposition, until, with the aid of the conclusive researches of Koch made known in 1882, we may assert that it is now firmly established. *We now teach that every lesion excited by the bacillus Kochii is tuberculous.*

**Etiology.**—The fact that almost one-seventh of the entire mortality of the human race is the result of tuberculosis, invests the etiology of the disease with great importance. It attacks all races, both sexes, and all ages, and is present in all countries. It is common in warm-blooded animals, being most frequently found in bovines. The great importance of the recognition of this statement lies in the fact that this class of animals supplies most of the meat and milk we consume as food.

There is quite a definite relationship existing between the number of people aggregated within narrow limits, as in densely populated cities, and the mortality from tuberculosis. The disease is very common in the United States, especially in the eastern portion. It is common in the British Isles, and quite as frequent in many of the warmer countries of southern Europe and the West Indies. It is less frequently observed in the extreme North and South. Mountainous regions contain a small percentage of cases, and the same is true of most of the extended plains of high altitude, notably those of our West.

**SPREAD OF TUBERCULOSIS.** The spread of tuberculosis depends upon the presence of susceptible individuals, into whom the tubercle bacillus may find its way and work its ravages. The distribution of the bacillus is undoubtedly very extensive, judging from the universality of tuberculosis. Susceptibility to the disease must also be very great. We will first consider the manner of entrance of the bacillus, *i. e.*, the mode of infection, and then those conditions which go to create susceptibility. It has been shown that the tubercle bacillus gains entrance into the body

in a variety of ways. It is probably most frequently inhaled, less often taken into the digestive tract in food, and still more rarely finds entrance through the cutaneous surface. The sputum of persons suffering from pulmonary tuberculosis is the principal source of the infectious agent. Until quite recently little or no care has been exercised in destroying or safely disposing of the expectorated matters, consequently portions thereof have dried upon clothing, floors or pavements, being broken into minute particles, and finally with the dust with which it has become commingled, is wafted about by the wind, and thus is inhaled. This explains why tuberculosis is far more frequently found affecting the lungs or larynx. Tuberculosis of other organs of the body may result from direct infection, as in the various forms affecting the skin and sub-jacent tissues; or any primary tubercular focus within the body may constitute an infecting centre, from which a general miliary tuberculosis may result. The possibility of developing tuberculosis by means of dessicated and pulverized sputum from tuberculous patients was demonstrated by Tappeniner at the Pathological Institute in Berlin, animals subjected to the inhalations of such matters becoming in all instances tuberculous. It is stated by Osler that it has been fully proved that the expired air of tuberculous patients is not infectious. This I cannot believe, as carefully conducted experiments of my own have demonstrated the presence of the tubercle bacillus in the expired air, and in the intimate relationship especially of husband and wife, transference of the bacilli to a new soil seems a possibility. No more trustworthy evidence of the danger of infection from dried sputum has been furnished than the observations of Dr. Geddings, of Aiken, S. C., who reports infection of the colored men employed in the spring of the year to whip the carpets belonging to the hotels after the removal of the tubercular patients, who have lived upon them during the winter. Since the discovery of a specific bacillus by Koch, there is a tendency to consider the long array of conditions supposed to possess a causative relationship to tuberculosis, as acting as predisposing causes only, and the bacillus tuberculosis as the necessarily ever present exciting cause. Many are, however, unwilling to fully accept this new view of the subject, being loth to relegate to the position of mere predisposing agencies, every factor but the bacillus. It seems to be generally accepted that some pathological change is favorable, if not essential, to the lodgment and activity of the bacillus. It is indeed questionable if the bacillus tuberculosis can be received and made to multiply in normal tissues. Klein, in his work upon micro-organisms, in referring to septic and zymogenic bacteria, says that "they also occur in the body of man and animals wherever there is dead tissue, in which they grow well and copiously." Those pathological conditions which are most frequently observed to precede or initiate pulmonary tuberculosis are first, protracted catarrhal inflammations of the bronchi, and secondly, broncho-and croupous pneumonia.

Of all the factors favoring the occurrence of tuberculosis, none is as important as hereditary predisposition. In fully one-half of all persons suffering from pulmonary phthisis—the most frequent form of tuberculosis—we are able to discover an hereditary tendency. Cases of congenital tuberculosis are met but rarely. Direct transmission of the virus from parent to child is advocated by some, especially by Baumgarten, but satisfactory evidence of such transmission is not yet forthcoming.

Often one of the parents or one or more of the brothers and sisters are subjects of tuberculosis, or have died of the same; or one of the parents gives a history of cough and other symptoms of pulmonary phthisis which existed years ago; in this class of patients physical examination may still reveal an old latent lesion; or tuberculosis may be common in the family of one of the parents, the disease not manifesting itself in the latter at all, and yet sometimes in all the children. Maternal inheritance is the more frequent.

The interesting work by Dr. Reginald Thompson upon the hereditary influence of phthisis furnishes much instructive information upon this subject. Dr. Thompson recorded 80 families with consumptive parents having an aggregate of 385 children, 194 of whom became phthisical, the remaining 154 being apparently exempt.

Various hypotheses have been advanced to explain hereditary influence in this disease. It is contended by some that the virus is transmitted in the same manner as in syphilis, and that the early manifestations are scrofula or tuberculosis of the cerebral envelopes or of the peritoneum. A comparison of the course of the early developments of scrofula and syphilis indicates that while scrofula is favored by constitutional conditions, yet the manifestations are developed as well by a great variety of exciting causes; whereas in syphilis, there is a symmetry in early development, and an independence of exciting causes which marks the true blood disease.

Direct contagion from the mother subsequent to birth directly or by means of the milk has been suggested. Were this so, the constant association of the child with the mother would suggest greater and earlier results from the consumptive mother's influence than from a diseased father. But this is at variance with observations. Another hypothesis advocated by many considers that a favorable soil only is inherited, a soil favorable to the presence and activity of the specific bacillus. This view seems to be the one which finds most favor. It harmonizes with the older as well as with the most recent views of the nature of this disease. The very recent observations of Gärtner (1893) conclusively demonstrate the transmission of the bacillus tuberculosis from mother to fœtus in canary birds, mice, and rabbits. Baumgarten strongly maintains the direct transmission of the bacilli as the essential feature of heredity, and Birch-Hirschfeld has reported the tubercle bacilli in a seven months



fœtus and the placenta, removed by Cæsarean section, the mother being the subject of miliary tuberculosis. Tuberculous lesions were not present in the child. Several competent authorities have since corroborated these observations. The infrequency of infection of the fœtus does not present an insurmountable obstacle to the acceptance of the theory that heredity depends upon the direct transmission of the bacillus, for it must be confessed that we know little or nothing as to the susceptibility of the fœtus, especially to so chronic a disease as tuberculosis. It is further probable that the conditions favoring infection are more fully developed toward the termination of pregnancy. It is supposed that placental lesions, hæmorrhages, necrotic areas, epithelial defects in the villi of the chorion, etc., may favor the passage of the bacilli. An interesting feature is the frequency of liver lesions in congenital infection. This is probably explained by the fact that the umbilical vein is the entrance highway, thus conducting the invading organisms directly to the liver and to the right side of the heart.

**Histology of Tuberculous Lesions.**—The bacilli, and more especially the toxins elaborated by them, exert an irritant influence upon the tissues in which they are deposited, with the resulting development of a cellular structure in which both the fixed tissue cells and the leucocytes take part. These lesions are divisible into (1) small grayish granular bodies designated miliary tubercle; (2) inflammatory processes of a diffuse character; (3) ordinary inflammatory products, viz., serum, fibrin, pus, epithelium, granulation and connective tissue. Our knowledge concerning the development of tuberculous lesions has undergone considerable revision of late, and a statement of our present understanding of this subject may be prefaced by the comment that there is nothing peculiar to tubercle in the various cellular elements involved in the process, nor in the relation of the several elements to each other, as other growths of parasitic origin are known to possess the same elements and the same arrangement, notably the actinomycetes.

**MILIARY TUBERCLE.** The several elements entering into the formation of a miliary tubercle are (*a*) epithelioid cells; (*b*) leucocytes; (*c*) giant cells; (*d*) a reticulum; (*e*) the tubercle bacillus.

(*a*) *Epithelioid cells.* The rounded or polygonal cells forming such a large proportion of the average tubercle are the product of proliferation of the fixed tissue cells, especially those of the connective tissue, and the endothelium of the capillaries, such a stimulation to growth of the tissue cells being excited wherever the tubercle bacilli may be lodged by the lymphatic or blood streams.

(*b*) The *round or lymphoid elements* which are abundantly present in most nodular developments of tubercle, are now believed to be simply leucocytes which have migrated from the vessels of the infected region, accumulating in the periphery of the group of epithelioid cells. It is

believed, also, that these cells do not undergo multiplication, but increase by simple aggregation of the wandering elements.

(c) *Giant cells.* The giant cell is less constant than the preceding. This mammoth element is the result of cell growth without division. Some pathologists, however, believe it may result from fusion of several cells, which we think is very doubtful. They contain many nuclei. Their presence appears to be favored by a limited number of bacilli, for both their number and size is greater when but few bacilli can be detected; consequently giant cells are not numerous in miliary tubercles, but are large and plentiful in tuberculous glands, lupus, etc. Many of these cells constitute a part of the basement substance, being apparently continuous with this tissue. Others are free in the meshes of this fibrous stroma.

(d) A *reticulum* is usually visible, especially in the periphery of the tubercle. It has its origin in the connective tissue matrix, contains rounded or oval nuclei, and is finally granular.

(e) *The tubercle bacillus.* This organism, which has been fully described in a previous section, is found in the cells, and also in a free state between them. They are present in many of the epithelioid cells, and often in much larger numbers in the giant cells. Their numbers in the walls of tuberculous cavities and their contents is large, indeed they form quite a percentage of the degenerated tissue.

The bacilli are also freely present in all tuberculous tissue undergoing caseous degeneration. In the lesions of acute general tuberculosis, especially in the young, the bacilli are present in immense numbers, more especially in the lesions found in the lungs. Bacilli are often absent in old tuberculous processes, or at least may be undiscoverable in such tissue by means of our present staining methods.

According to the theory of Metschnikoff the leucocytes sally forth and oppose the invading bacilli and under favorable conditions destroy them, which theory (phagocytosis) may in part at least account for the arrest of tuberculous lesions.

The *degenerative changes occurring in tubercle* are essentially of the nature of a coagulation necrosis, which begins in the centre of the tubercle granulation. As a result of this change the cell elements lose their outlines, the nuclei gradually disappear, and a yellowish, cheesy, amorphous mass, which will no longer respond to stains, is the result. In conjunction with this caseous transformation a sclerotic process is progressing, especially in the outer zones of the tubercle, but which is not equally prominent in all. This increase of the fibrous element results in the development of firm granulations, some possessing a high degree of consistency. Others may be transformed into connective tissue nodules.

Examinations of many of these granular bodies, developing as the

result of the irritant action of the bacilli, will reveal quite a *variety in structure*. In some the granulation cells, in others granular matter will be predominant; again, epithelioid elements will preponderate; and a certain proportion will contain degenerated visceral elements, pus, etc.

In their growth tubercular lesions invade and destroy the tissues, especially the connective tissues; they may project from free surfaces, and if involving a cavity of small size, it may become filled. If the bacilli gain access to the blood or lymphatic elements there is extensive sowing of the infective agent, and a widespread eruption of tubercles.

*Inflammatory processes of a diffuse character, the products undergoing cheesy degeneration.* The true nature of this variety of lesion, which was correctly taught by Laennec to be of a tuberculous nature, but which position was long antagonized by the German school of pathologists (especially by Virchow), has been definitely determined by means of bacteriological methods. It is present in the form of a diffuse cheesy infiltration of the kidneys, testicles, bladder, prostate, etc.; also in the brain in the form of extensive tuberculous masses, or, as observed recently in a man suffering from active pulmonary phthisis, in a rapid local tubercular inflammation of the brain, quickly terminating in caseous degeneration and diffuence; or, as growths of large area but without much thickness, upon the serous membranes, most frequently upon the peritoneum. These lesions are rich in round cells and tend to the rapid development of caseation. Miliary growths may be detected scattered through the lesions prior to their obscuration by the rapidly progressing coagulation necrosis. It is probable that these diffuse lesions always develop from numerous microscopic foci, in which fully developed nodular tubercles are present, the intermediate structure becoming gradually filled with cell products and inflammatory exudate, *e. g.*, in the case of the pulmonary tissue the air vesicles in the neighborhood of the primitive foci contain proliferated epithelium, a few leucocytes; and if the process is a rapid one, variable amounts of serum, fibrin, and pus cells. The walls of the alveoli are infiltrated with round cells. Caseation is first apparent at the centres of the small nodules, but ultimately involves areas varying in size from a few lobules to an entire lung. As studied in solid organs, this process does not differ in essence from the above.

Suppuration occurring in association with tuberculous lesions is generally held to be the result of a mixed infection, although in many collections, apparently purulent, there is an absence of the organisms upon which suppuration is dependent, the collection being composed simply of the *debris* of tubercle; also inoculation experiments with the tubercle bacillus, and especially its products, have demonstrated their ability to excite suppuration unaided by accessory germs.



## MILIARY OR ACUTE GENERAL TUBERCULOSIS.

By general tuberculosis we understand a widely diffused eruption of miliary tubercles in the tissues, and running a rapid course. Occurring thus, the tubercles are found most frequently, and in the largest numbers, in the lungs, pleuræ, peritoneum, lymphatic glands, spleen, liver, kidneys and cerebral membranes; and less frequently in the choroid coat of the eye, the marrow of bones, the thyroid gland and the heart. There is no relative frequency with which these several parts are invaded. In a given case, it may even happen that there is an extensive development of tubercles in one organ or tissue, while all other parts remain healthy. The symptoms of this disease also present the greatest variations, as can be readily surmised from the number of organs involved. Still, symptoms indicative of local deposition of the tubercle are not always present. The disease very often, indeed, for a time at least, gives evidence only of general disturbance of health. In such cases, great difficulty in separating the affection from other acute infectious diseases is encountered. When the eruption of tubercles is in the lungs or brain, definite and characteristic symptoms are generally observed.

Acute miliary tuberculosis may occur at any age from infancy to advanced senility. It arises always from infection with the tubercle bacillus. Why, in these cases, the symptoms should develop so rapidly and follow such a malignant course, is a mystery. Gilman Thompson quotes Reich, who reports an epidemic of acute tuberculosis in infants caused by a tuberculous midwife, who resuscitated still-born infants by applying her mouth to theirs and breathing into their lungs. The disease has also arisen from infection acquired during the Jewish rite of circumcision.

Cases have occurred in which the symptoms appeared suddenly without apparent cause. Buhl has proven most conclusively that such cases arise from a general tuberculous infection resulting from an auto-infection from a cheesy focus somewhere in the body, the presence of such a localized tuberculous deposit having been hitherto unsuspected. It is presumed that tuberculous glands ulcerate into veins, and thus produce rapid systemic infection. Osler concurs in this view, and says that auto-infection arises in this way also from the lungs, bones and kidneys. More frequently, however, the acute infection is secondary to *ascertainable* disease in the lungs and other tissues above named. The onset of apparently idiopathic cases may be attributed in some cases to tuberculosis of the thoracic duct, which thus becomes a central point for the wide dissemination of the tubercle bacilli. Weigert discovered the important fact that the veins, more especially the pulmonary veins, are occasionally tuberculous. This process is nearly always secondary

to tuberculosis in surrounding structures, the growth first invading the venous wall from without, and extending thence into the lumen of the vessel, thus affording an admirable opportunity for general infection through the agency of the blood current. Acute miliary tuberculosis occasionally results from the primary infection of the blood by the inoculation of bacilli. The disease then rapidly becomes general, involving one or many organs.

**Clinical Varieties of Acute Tuberculosis.**—Miliary tuberculosis presents the greatest varieties in its clinical manifestations. Several forms have been recognized, these being determined in a measure by the special localization of the tuberculous deposits.

**TYPHOID FORM; MILIARY TUBERCULOSIS, IN WHICH THE SYMPTOMS ARE THOSE OF GENERAL INFECTION.** As the term indicates, this variety presents a great resemblance to typhoid fever. Its onset is usually characterized by a prodromic period represented by anorexia, weakness, feverishness, etc. There next appear a marked rise in temperature, cough (which is usually dry, but may be attended by muco-purulent expectoration), dry tongue, rapid and feeble pulse, and delirium with flushed face. The temperature range in miliary tuberculosis is characterized by its irregularity. The maximum daily temperatures vary between 103° F. and 107° F. The remissions are very great, the temperature being even subnormal in one part of the day, while hyperpyrexia is present later. The highest temperature is usually observed in the evening. Exceptionally, the reverse obtains, and the morning temperature is the higher. It is said that this inverse type of temperature is present far more frequently in acute tuberculosis than in any other disease. The causes which determine the height of the fever can hardly be said to be known. The temperature is higher when the serous membranes are affected. The degree of fever also seems to depend upon the severity of the infection of the blood rather than upon the localization of the disease in any particular organ. Reinhold has reported cases which have run an afebrile course. I have seen the same, and have corroborated the diagnosis by post-mortem examination. The respirations are nearly always affected. At first they are simply rapid and of normal depth. Later they become shallow. Dyspnœa may or may not be present.

With the progress of the disease the patient emaciates, the skin becomes very pale, and in the latest stages cyanotic. Diarrhœa, with distended abdomen, is not uncommon; though it is otherwise stated by many authors. The spleen is usually enlarged. Jaundice is occasionally observed. The tongue is dry and brown. Sweating is often uncontrollable, and accompanied by sudamina. Urinary examination discovers albuminuria and often peptonuria. Nervous symptoms finally become important clinical phenomena. Delirium and even acute mania are observed, particularly at night. In general, however, the patient is dull, and develops usually a gradually increasing coma.

The duration of the disease is very variable. The patient may succumb within ten days or two weeks, or resist its ravages for six weeks or longer. With the approach of a fatal termination, there is apt to be a fall of the temperature to a subnormal point, or, if it is already low, a sudden elevation may occur. The patient now develops marked cerebral or pulmonary symptoms, *i.e.*, a progressive coma, with perhaps rigidity of the neck and paralysis of the ocular muscles, or evidences of pulmonary œdema.

Prior to the appearance of the symptoms of local disease the resemblance to typhoid fever is often confusing. *As contrasted with typhoid fever* miliary tuberculosis presents a more irregular temperature range, the respirations are more frequent, bronchitis more marked, and defective oxygenation of the blood more common; and the spleen not as early nor as constantly enlarged. Leucocytosis can be present in tuberculosis; it is absent in typhoid fever. Epistaxis may be present in both affections, but is less frequent in tuberculosis. The characteristic eruption of typhoid fever is absent in the disease under consideration. Ehrlich's diazo reaction is of little value, as it is present in both diseases. In doubtful cases, the ophthalmoscope may be appealed to for the discovery of tubercles in the choroid. Inasmuch as this tissue is one of the less frequent spots for tubercular deposits, a negative result can possess but little value. The blood may be examined for bacilli. Typhoid bacilli may be discovered in the blood of the spleen, which organ may in important cases be punctured and the withdrawn blood subjected to bacteriological examination.

**PULMONARY FORM; MILIARY TUBERCULOSIS IN WHICH SYMPTOMS RELATING TO THE LUNGS ARE MOST PROMINENT.** This variety may develop rapidly or slowly. It is often grafted on a pre-existing pulmonary lesion, which may or may not be tubercular in character. More often the patient is known to have been a subject of chronic pulmonary phthisis for a long period of time. In children whooping cough, measles, and other infectious diseases may precede. When the development is acute, the symptoms often suggest croupous pneumonia or a severe general bronchitis; indeed, the latter condition is the usual mode of onset in this affection. The attending cough is usually troublesome, and the expectoration muco-purulent or rusty. The respirations are much quickened, often 50 or 60, and even higher, especially in children. The degree of dyspnoea is not accounted for by the physical condition of the lungs. Cyanosis soon becomes a prominent symptom, and continues to the end. Hæmoptysis is occasional. Physical examination usually reveals the evidences of a severe general bronchitis. Signs of involvement of the finer tubes are generally present. Localized consolidation of the lungs can be frequently discovered, especially posteriorly in the bases. Some authors speak of an abnormal resonance which is probably due to acute emphysema. The anterior surface of the chest often presents



a normal percussion sound, and only a few râles are observed on auscultation. Cases will be observed in which physical examination gives only negative results. Post-mortem examinations in such cases show that the tubercles do not involve or compress the air cells, or are not close enough together to modify the physical signs. The spleen is usually enlarged and the liver may be swollen or tender. The abdomen is frequently tympanitic. Digestion is feeble and vomiting frequent. Cerebral symptoms may develop late.

**Diagnosis.**—In addition to a consideration of the facts already stated, in doubtful cases, the sputum should be carefully examined for bacilli. Careful search for old tubercular foci must not be neglected. The setting in of symptoms of tubercular meningitis, which occasionally occurs, is of inestimable diagnostic value.

As to local pulmonary symptoms, the most important are those of a diffuse bronchitis, accompanied by a pronounced dyspnoea and cyanosis.

**Prognosis.**—The duration of acute pulmonary tuberculosis is very variable. Rapidly developing cases are often fatal in two weeks. Others may last for several months. The causes of death are exhaustion from the fever or the systemic infection, pulmonary œdema, and heart-failure. Acute miliary tuberculosis is uniformly fatal. Cases reported as recovered are open to the objection of being examples of mistaken diagnosis.

**Treatment.**—To specify a course of treatment for a disease as hopeless as miliary tuberculosis, seems almost useless. One can only treat the patient on general principles, relieving symptoms as they arise, and lessening suffering. Remedies undoubtedly exert a specific effect in this direction, although not curing. Cases reported as cures are almost certainly not examples of acute tuberculosis. The remedies from which most may be hoped are *iodine*, *phosphorus*, *arsenicum*, *sulphur*, *hyoscyamus*, *stramonium*, *china*, *ferrum*, *rhus tox.*, *calcarea carb.*, *phos.*, and *iod.*, and *lycopodium*.

THE MENINGEAL VARIETY OR THAT FORM OF TUBERCULOSIS IN WHICH MENINGITIS IS ASSOCIATED WITH A DEVELOPMENT OF TUBERCLES IN THE MEMBRANES OF THE BRAIN, THE INFLAMMATORY CHANGES BEING PROBABLY SECONDARY TO THE GROWTH OF THE TUBERCLES. Such a meningeal tuberculosis is, in fully one-half of the cases, a part only of a general miliary tuberculosis; but the marked character of the cerebral symptoms usually prevents a proper appreciation of the general character of the disease. This affection is sometimes designated acute hydrocephalus, or, on account of the special involvement of the membranes at the base of the brain, basilar meningitis. As a separate clinical entity it was first described by Dr. Robert Whytt, in 1768, he giving it the name of acute hydrocephalus, for reasons which will appear presently. This name clung to it until very recent years, notwithstanding that its true pathological character was discovered by Papavoine, as long ago as 1830. It is a disease of comparative frequency and of considerable importance.

**Morbid Anatomy.**—Collections of fluid in the ventricles and tissue softening were observed long prior to the discovery of the tubercular lesion at the base of the brain, and this led to the old term, acute hydrocephalus. The tubercles are developed mainly in the pia mater. While they may be found on the surface of the brain, or in the ventricles, it is especially at the base of the brain that the granulations are found in largest numbers. The pia mater becomes the seat of inflammatory changes, excited by the tubercular process. Some degree of inflammation of the underlying cerebral tissue is always associated, although the development of the tubercle granulations prior to the meningeal inflammation is inferred rather than proven. The exudate varies in character, being serous, sero-fibrinous, or sero-purulent. The amount of exudate bears a greater relationship to the intensity of the meningeal inflammation than to the number of tubercles present. At times the exudate may possess decided consistency, matting the structures together. The base of the brain and the nerve trunks are bathed in the exudate, the lateral ventricles may be distended, and the fluid may extend *via* the Sylvian fissure to the lateral and less frequently to the superior surface of the brain. According to Cornil, the tubercles are developed especially in the walls of the small bloodvessels. As the result of their presense and of hyperplasia of the tunica adventitia, the vascular lumen is encroached upon and the circulation more or less obstructed, leading to œdema, minute hæmorrhagic foci and softening of areas of cerebral substance. The tubercles are found in largest numbers in and about the Sylvian fissures and the optic chasm. They may be so few as to be difficult to discover, or the pia may be studded with them. When there is doubt, the membrane should be removed from the perforated spaces and inspected, or the middle cerebral artery should be dissected out and examined with a low power. A glass with a dark ground is most suitable upon which to spread the specimen. The tubercles appear as minute translucent granules. Those which have undergone caseation appear yellowish and opaque; agglomeration of numbers of granules forms masses of considerable size. In most autopsies a general miliary tuberculosis is demonstrated to have taken place. If the patient is young, the organs most likely to present extensive infection are the bronchial glands and the peritoneum; in adolescents and adults, the lungs.

**Etiology.**—The etiology of tubercular meningitis is essentially that of miliary tuberculosis generally, which has already been considered at length and need not be repeated at this place. It is questionable if a primary tuberculosis of the cerebral meninges ever occurs, although such theories as the passage of bacilli through the cribriform plate of the ethmoid bone have been advanced for unexplained cases. The location of tuberculous foci in the body may be so various and widely distributed

that it is easy to overlook one which has not excited symptoms. More carefully made autopsies would clear up many cases of doubtful etiology. According to Jacobi, tubercular meningitis is more frequent during the first year than has generally been supposed. This is due to the different symptomatologies at various periods of life. Most cases occur between the second and the fifth or seventh years. Later than this the disease is not frequent, being then usually a late manifestation of chronic phthisis. Hereditary feebleness of tissue-nutrition predisposes, as in all forms of tuberculosis; and infection by means of food, especially of milk, to which young children are especially exposed, is probably the exciting cause of many. More than one case is frequently observed in the same family, suggesting an hereditary proclivity. Certain factors seem to act as predisposing agents. Males are more frequently affected than females. Children, especially those between the ages of one and five years, are the victims in the majority of cases. Fagge, notwithstanding the unanimity of other authors respecting this latter point, is not prepared to lay down any definite relation between the tubercular meningitis and the age of its victims, inasmuch as the statistics of Guy's Hospital show that all ages are about equally affected. Several observers (Baumler, Uffelmann and others) have reported cases of tubercular meningitis as occurring subsequently to erythema nodosum. The embolic origin of this disease, and its ability to develop embolic processes, suggest a possible mode of infection of the meninges.

Measles, whooping cough or some other infectious disease has frequently been observed to precede the tubercular meningitis. The relationship is doubtful, but they possess the peculiarity of often arousing to activity any dormant constitutional vice. There can be no doubt, when circumstances are favorable, that neuropathic constitutions and traumatism are predisposing factors.

**Clinical Course.**—The progress of tubercular meningitis is variable. Prodromes may be marked and protracted, or almost absent. The inflammation may appear suddenly and with great intensity, or so slowly and be so protracted as to be classed as a chronic affection. Between these extremes there are many gradations. Authors recognize several stages of the disease, which, however, are not distinctly defined, but merge gradually one into the other. In the typical form, as it appears in young children, there is a history of general bad feeling for some days, or even weeks, previous to the onset of the disease. There may be a history of a fall or of recent convalescence from an acute infectious disease. The appetite is poor, the patient is irritable, and frequently manifests a change in disposition. The mental state is often evidenced by a state of apathy or indifference. Sleep is disturbed at night, and there may be drowsiness during the day, and there is often a slight cough. There is always more or less hyperæsthesia of the



senses. Slight headache is likewise present. These premonitory symptoms last for a variable period—for a fortnight, it is usually stated; but they may continue for weeks before the true nature of the malady is asserted. The onset is marked by a group of interesting symptoms. There may be a convulsion or chill; but usually the prodromic symptoms gradually increase, and finally ultimate in headache, vomiting, irregularity of respiration and pulse, and fever. Vomiting is the most constant of these. Nausea is not common, and the vomiting effort is slight. Continuance of these symptoms during the entire duration of the disease is not uncommon, although they usually cease within a few days. Intermissions are common, a cessation for a day or two being rarely followed by a return. Eating, drinking and motion excite it.

**RESPIRATION AND PULSE.** Both the respiration and pulse are at first accelerated; but in a few days, in very acute cases, they present the characteristic slowness and irregularity. Often there is marked variability in the pulse and breathing, even within a few minutes. In suspicious cases these should be carefully observed while the patient is asleep, as in the early stages of the disease these symptoms can only be observed at such times; later they may be constant. The breathing is frequently sighing. The temperature presents no especially characteristic curve. It is usually increased, especially in the evening, the maximum being in the neighborhood of  $103^{\circ}$  F., with the exception of just before death, when it sometimes rises to as high as  $107^{\circ}$  F. Some cases may run their course with a subnormal temperature. The pulse does not increase in frequency proportionate to the temperature.

**PAIN.** Headache is often severe, and inclined to be paroxysmal. It is usually frontal, and attended by sensitiveness to light and sound, and contracted pupils. The patient is inclined to compress the forehead and bury the head in the pillow.

**NERVOUS AND MENTAL SYMPTOMS.** Patients suffering from tubercular meningitis seem disinclined to be disturbed; they are easily annoyed. Questions receive halting, but intelligent, answers. The face is flushed, and the expression is one of anxiety. There may be mild delirium, and sleep is disturbed in some cases by the hydrocephalic cry and grinding of the teeth. Muscular rigidity of the abdominal walls, of the spinal muscles, clenching of the hands, muscular tremors and twitchings, strabismus, ptosis, etc., are symptoms often present, and due to cerebral irritation.

As pressure upon the brain increases, due to the accumulating exudate, the foregoing symptoms of irritation are overshadowed by those due to pressure. The transition to unconsciousness, which is the most important symptom of pressure, may be rapid, but is not usually so. For a time, periods of great restlessness and suffering alternate with periods of stupor. The muscles of the posterior portion of the neck become

rigid, the head is retracted, and may be rolled from side to side. The pulse and respiration changes described become much more pronounced and the Cheyne-Stokes respiration may be present; the mouth is covered with sordes, food is taken only when forced, the bowels are usually constipated, and the contracted abdominal walls cause a deeply concave or "boat-shaped" abdomen. The pupils are now dilated, the eye is congested, and a muco-purulent secretion bathes the balls and collects upon the edges of the eyelids. Ophthalmoscopic examination of the fundus may show a developing neuro-retinitis, and in a minority of cases tubercular granulations develop on the choroid. A peculiar vaso-motor disturbance described by Trousseau as *tache cerebrale*, consists of a gradually developing and slowly fading red line produced by drawing the finger-nail over the skin, preferably upon the inner surface of the thigh or across the abdomen. Much has been made of this symptom, but it is now generally regarded as of no diagnostic value. Small areas of congestion appear for a short time on the skin of the face, forehead and ears, and present a brilliant contrast to the pale background. There is usually incontinence of urine and fæces. In the late or paralytic stage there is increasing coma, failing circulation, with very slow or rapid and feeble pulse, and varying degrees of paralysis, both in extent and completeness. Contractions of the muscles of the jaw and neck and sometimes of the back occur; also spasms, rigidity or paralyses of the limbs of one side. It is not rare at this time for typhoid symptoms to appear; constipation gives place to diarrhoea, with distended abdomen. Before death, which occurs in coma or convulsion, the temperature may rise to a very high point or sink several degrees below normal (94° F.).

**Analysis of Symptoms.**—The fully developed disease depends for its phenomena entirely upon the situation of the pathological process. As already stated, this is usually at the base of the brain; occasionally, it is over the convexity. In the latter instance, epileptiform convulsions of the Jacksonian type, and other evidences of cortical disease, are prominent features of the case. In the former, quite a variety of symptoms appear. Explosive vomiting, so characteristic of basilar disease, is among the first symptoms. It may take place immediately after eating; or the vomited matters may consist of bilious material. It lasts usually for from a few days to a week. When it disappears for a period of twenty-four hours, it is not likely to be renewed. As more marked nervous phenomena, we have convulsions, paralyses, and unconsciousness as initial symptoms. The convulsive seizures are epileptiform in type, and may be local or general. Their duration is variable. They may last for hours, or again but for a few minutes. The hemiplegia is of gradual onset. The attack of unconsciousness is sudden and temporary, taking the place of a convulsion, as it were. The disease now being fully developed, the headache increases in intensity, until it is of agoniz-

ing severity. The child gives evidence of its suffering from this cause by clutching first at one side of the head and then at the other. From time to time, it gives forth a piercing shriek, the latter being a symptom considered as characteristic by Trousseau, and dignified by him by the special name *cri hydrocephalique*, or the hydrocephalic cry. Emaciation increases rapidly. The mental faculties become more and more dulled, until stupor is profound. The pupils respond sluggishly to light. Not infrequently, one pupil is larger than the other. Other eye symptoms are noted. Strabismus is a frequent phenomenon, and nystagmus is occasionally observed. Other symptoms are vertigo, facial twitching, and tetanic rigidity. As the patient approaches his end, unconsciousness becomes absolute. The rigidity of the neck increases, and the head is drawn backward. The pupils become widely dilated. The patient grinds his teeth. Paralyzes of various cranial nerves, especially those of the eyeball and the face, set in.

Throughout the disease, certain irregularities in the circulation, in the shape of morbid flushing and paling, are observed. The abdominal walls are markedly retracted. Constipation is almost invariably obstinate. The tongue is usually red and dry. Towards the end, defecation and micturition become involuntary. A prelethal symptom, which often leads to delusive hopes of recovery, is a return of consciousness.

Tubercular meningitis in the adult presents some few variations from the type observed in childhood, as above described. Prodromal symptoms are more rare. Any ill health is usually ascribed to pulmonary involvement. The cerebral symptoms themselves are more insidious and of slower development. Delirium is an early and persistent phenomenon. Rarely the first external evidence of the disease is a convulsion, which is followed in a day or two by death. It is important to note that tubercular meningitis in the adult has often been confounded with hysteria.

The usual duration of tubercular meningitis is from one to three weeks. Rilliet places the limits at from twenty to thirty days. It is probable, however, that this disease may exhibit all shades of acuteness and chronicity.

**Diagnosis.**—The symptoms presenting no characteristic differences from other varieties of meningitis affecting the same localities, one is obliged to rely mainly upon attending phenomena for diagnosis. Meningitis at the base is, in the young, practically always tubercular; that of the convexity is scarcely ever so. The association with meningitic symptoms of tubercular disease in other organs is strong presumptive evidence of the tubercular nature of the cerebral disease, also, the absence of a history of the operation of causes of a nature to induce suppurative meningitis, such as injuries, otitis, etc., and by the longer duration of the tuberculous variety as compared with suppurative meningitis. The



occurrence of paralysis of cranial nerves is also suggestive, especially as indicating a basal inflammation. The presence of tubercles in the chorioid is valuable evidence, but their absence is not of much negative value. Retinal changes which were at one time looked upon as significant have been shown to occur so frequently in other affections of the brain, as to be of little diagnostic value. Typhoid fever is without question liable to be confounded with tubercular meningitis. There is no special constantly present symptom or condition, of which I have knowledge, that will prevent error. One must be guided entirely by the totality of the symptoms in each case in order to reach a correct conclusion. The difficulties here are enhanced by the occasional complication of typhoid fever by meningitis, which seems to be a special sequential condition, for typhoid bacilli have been found in the meningeal lesions. It is taught, and is true in many instances, that the examination of the knee-jerks will afford some differential aid. In typhoid fever these are either normal or somewhat exaggerated. In many cases of meningitis they are absent. Sometimes they are exaggerated.

Cerebral syphilis presents many of the phenomena of tubercular meningitis. A presumptive diagnosis is here made by the age of the patient; in tubercular meningitis, the patient is usually a child; in cerebral syphilis, a young or middle-aged adult.

Persistent pain in the head with fever, when occurring in a young child, should always excite suspicion of meningitis, but as there are other causes for this group of symptoms, search for such must be made. It seems only necessary to call attention to enteric fever, otitis, bronchitis, pneumonia, gastro-enteric troubles, and the spurious hydrocephalus, which most frequently excite symptoms resembling those of meningitis, but a careful consideration of the history of the case, coupled with a thorough physical examination, will often aid to a differentiation.

**Prognosis.**—This disease is universally acknowledged to be always fatal. And yet there is hardly an able clinician anywhere who has not seen cases in which the evidence of tubercular meningitis was complete, make most excellent recoveries. Such cases are then looked upon as examples of diagnostic error. It is believed that the majority of them are instances of congenital syphilis. I can recall one case in my own practice in which the evidence seemed complete, and in which incomplete recovery ensued. All active symptoms subsided, but left the child a mental wreck. It died two years later of tubercular trouble elsewhere.

**Treatment.**—Children of tubercular parents may possibly be preserved by the early adoption of measures calculated to antagonize the strumous cachexia. Physical development should be favored and mental growth inhibited. The unfavorable outlook in these cases should not prevent a lack of enthusiasm in our efforts for the recovery of the

patient. Cold to the head is a useful measure in the acute cases. It is not so much the influence of the cold *per se* that is needed as the reflex influence of its application. For this reason, cold water, and not ice, is better. Absolute rest is necessary. Rest the patient completely, both body and mind. Darken the room; keep out noises; disturb him as little as possible by efficacious attention. The diet must be adapted to sustaining strength, and must be at the same time digestible. Well-made beef-broths and milk fill every indication for food.

The resort to operative treatment is well worth consideration. There is at present no precedent for its application. We do know, however, that tubercular disease of the peritoneum has been cured by abdominal section. Analogy would lead us to expect at least a favorable result by opening the meningeal cavity. This recommendation is based largely on theoretical considerations. In putting it into practice the physician should be guided entirely by his own feelings as to what he would do under the circumstances were the patient of his own family.

Among remedies, *iodoform* stands at the head. This drug has, in several instances (I have twice observed it do so) produced symptoms indistinguishable from meningitis. It is customary with old-school physicians to shave the scalp and apply an iodoform ointment for two or three days. The internal administration of the drug will probably do as much, if not more, good. I have used it in the second decimal trituration, giving one tablet of the same every two hours.

*Apis* is another remedy whose symptomatology bears a close resemblance to tubercular meningitis. It exhibits the fidgety restlessness of the beginning of the disease, the loud hydrocephalic cry. The child bores its head into the pillow, rolling it from side to side. Convulsions may be present, or the child may be convulsed on one side and paralyzed on the other. Eye symptoms in the shape of strabismus appear. Spasm of individual muscles, especially of the flexors, may be present. The symptoms of this drug are marked, therefore, by irritability.

*Helleborus*, on the other hand, is adapted to cases in which apathy is the prominent feature. The onset of exudation does not contraindicate it. There are shooting pains in the head; the child bores its head backward into the pillow; the head is hot; the forehead, wrinkled; automatic motions of one arm and one leg; eyeballs drawn upward; face flushes up suddenly and gradually pales; pupils do not react to light; corrugation of the muscles of the forehead, with more or less constant chewing motion of the mouth.

*Iodine* has been lauded by Jousset, and has pathological considerations in its favor.

*Sulphur* is indicated mainly by reason of its adaptability to constitutional dyscrasiæ.

Hughes recommends *belladonna*, *bryonia*, *helleborus* and *sulphur*, plac-

ing his main reliance, however, on the former two. *Belladonna*, he says, continues to be the proper remedy as long as effusion keeps off, when *bryonia* steps in. Farrington observes very properly that this remedy bears only a superficial resemblance to tubercular meningitis. Its genius adapts it to sthenic conditions, which do not characterize the disease under consideration.

The recommendation of *apocynum* for the effusion is without good reason, pathological or otherwise. Such remedies as *digitalis* (Baehr), *bryonia* and *apis* will prove better

*Calcareæ ostrearum* and *phosphorica* are remedies indicated like *sulphur*, because of their constitutional states rather than by their symptoms, and they should prove of service in some cases. They are highly praised by Jahr. A strumous child of four years, parents phthisical, was the second child of the family to be attacked with tubercular meningitis, an older sister having died of that disease two years previously. The onset followed a rather protracted prodromic period. The disease reached the stage of effusion, and presented convulsions with paralysis of the right side as well as of some of the ocular muscles. The hydrocephalic cry was well marked, the abdominal walls retracted, and the bowels constipated. *Lycopodium* 6x prescribed upon a number of its well-known symptoms was followed by gradual relief of the symptoms. The patient made a tedious recovery. I have several times given this medicine with good results to strumous children who seemed to be developing symptoms suggestive of tubercular meningitis.

Several of the medicines of value are considered under the head of acute leptomeningitis, which article should be studied in conjunction with the present one.

### TUBERCULOSIS OF THE LYMPHATIC GLANDS. (SCROFULA.)

For many years scrofula was regarded as a distinct disease, but is now known to be but one of the numerous manifestations of tuberculosis. Schuppel, in 1871, demonstrated the tuberculous character of the lesions. Later the researches of Koch carried general conviction as to the identity of the two processes, scrofula and tuberculosis. The term scrofula is used to denote a condition characterized by enlargement of the lymphatic glands, associated in many cases with sluggish inflammatory affections of the skin, mucous membranes, bones, joints, etc. Since the discovery of the tubercle bacillus in scrofulous glands, many pathologists have assumed the tubercular nature of all so-called "scrofulous swellings." It cannot, however, be said to have been positively determined that all of these cases are of this nature. Delafield and Prudden state "that in a considerable number of so-called scrofulous inflammations of the lymph nodes, there is no formation of tubercle tissue, and we find no tubercle



bacilli, so that we consider this class of cases as simply inflammatory with a tendency to cheesy degeneration." Personally, I have repeatedly failed to discover tubercle bacilli in well-marked cases of "scrofulous" adenitis.

The tardy course and mild nature of tuberculous adenitis has led to speculation as to whether the bacillus of tuberculosis operates with the same intensity in the various organs in which it is found. Lingard has made some interesting experiments to determine this point, and these prove conclusively that the bacillus as found in the lymphatic glands is not nearly as virulent as when obtained from other locations. Inoculations with the latter have been shown to lead to the development of tuberculosis in one-half the period required by the former. The subsequent duration of life in the subjects thus rendered tuberculous was relatively the same. This experimenter further observed that the infectious principle obtained from the lymph glands became progressively more virulent, as it was passed from one guinea-pig to another, thus indicating that the character of the infection is the same in all locations, but differing only in virulence, that of the lymph glands representing, perhaps, the lowest potency. Arloing in his experiments showed that inoculation of guinea-pigs with the product of scrofulous glands produced tuberculosis; while inoculation of rabbits gave negative results. Eve, repeating Arloing's experiments, found that inoculations of rabbits and guinea-pigs with scrofulous glands produced tuberculosis in both animals, the disease in the former, however, running a less acute course.

In tuberculous glands we find inflammatory changes, either diffused or confined to the periphery of the tubercle granulations, and this inflammation stimulates these miliary tubercles to increased growth. The latter, as in other locations, consist of numerous small lymphoid cells, larger polyhedral cells, and usually giant cells. Cheesy changes occur in the centre of these tubercles, and these present the same appearances as when occurring in other locations. If the process is rapid, suppuration occurs. If chronic, there is a marked thickening of the reticular tissues as well as of the capsular; or the caseated matters may soften and liquefy and be evacuated. The tendency to suppuration is marked, especially in the cervical glands. Only exceptionally may tubercle bacilli be found in the pus. Occasionally the cheesy substance is surrounded by a wall of firm connective tissue, constituting a distinct capsule. The enclosed material may calcify or soften, thus forming a cyst. This barrier developed between the infectious material and the organism is an interesting illustration of the conservative processes of nature in the conflict which is waged between the cells of the tissues and the invader—the bacillus tuberculosis—a conflict in which the tissues are victorious.

The principal objection to the belief in the tuberculous origin of

scrofula is found in the absence of bacilli in many cases. The mere microscopical examination of the diseased glands counts for nothing, unless inoculation experiments have likewise been performed.

Coming now to etiological considerations, we find tubercular adenitis especially liable to occur in children, although the number of well-developed cases I have observed in adults has surprised me. At the time of writing, two cases are under treatment. One a man of forty-three, and the other a woman sixty-four years of age. The woman has heretofore been free from evidences of tuberculous disease, the man has very slight signs of disease of the apex of the right lung, although there is no evidence of activity of the pulmonary lesion. There is every probability of the infection of the woman by a tuberculous husband, through the medium of a wound. The man has a very bad family history.

The habitual or frequent neglect of hygienic rules forms an important predisposing cause of tuberculosis of the lymphatics. Children who are confined most of the time in dark, damp, poorly ventilated dwellings, and at the same time are fed on coarse farinaceous food, are especially predisposed.

The influence of heredity in the transmission of tubercular adenitis is as yet somewhat of a mooted point. It has been observed that children born of parents actively phthisical are often affected. In such cases it is not necessary to appeal to a direct hereditary transmission in finding a cause for the disease. Children born of such parents are necessarily in possession of a comparatively limited health capital, and have correspondingly feeble powers of resisting the inroads of disease. Let such a child be seized with a catarrhal rhinitis; the agents of dissemination of the disease—bacilli from the tuberculous parents—are in the household, and infection takes place.

The discovery of the tubercular origin of scrofula has deprived the latter of many of its essential symptoms, leaving the adenitis as the only phenomenon to keep alive the term as a clinical entity. The catarrhs, the skin diseases, etc., act simply as foci of irritation of the lymphatic nodes, which are then rendered suitable for the reception and propagation of the bacilli.

Tubercular adenitis may be local or general in its development—much oftener the former. The local developments occur most frequently in the neck, the chest or the abdomen. Involvement of the cervical glands has attracted most attention in the past, especially on account of the superficial and conspicuous character of the lesion. Disease of the deeper-seated glands, the bronchial and mesenteric, is present much oftener than supposed, as the condition is readily overlooked. The fact that lymphatic glands which have undergone tuberculous change are a constant menace to the individual, and that the majority of cases of military tuberculosis owe their origin to infection derived from this source, has already been suggested.

**CERVICAL FORM.** This variety is most frequently observed in children. It is very often associated with disease of the mucous membranes of the upper respiratory tract, with otitis media, or with affections of the cutaneous surface of the head and neck. The relationship between these symptoms and the glandular enlargements has already been explained. The glandular tumors present a smooth surface, the overlying skin generally being normal in appearance, and movable. Later, inflammatory symptoms become active and the skin grows adherent. If the disease remains confined to the cervical region, recovery is usual. Extension to the axillary glands or even to more distant groups, increases the gravity of the case materially. A tubercular pleurisy or pulmonary tuberculosis may follow an extension to the lower cervical or axillary glands. Many children with tubercular adenitis later develop a fatal broncho-pneumonia. The general health of the patients varies much. There may be fever in very acute cases, but the temperature is more apt to be normal. Muscular feebleness with a tendency to sweat readily is common. The appetite is often fickle; pale skin and the general evidences of anæmia are often present.

**BRONCHIAL FORM.** Recent researches have shown the great frequency of involvement of the glands situated in the mediastinum. When we consider the ordinary mode of infection with the tubercle bacillus, *i. e.*, by inhalation of the dried organism, and the relationship of the lymph-glands about the bronchial tubes to the lungs, this frequency will not seem strange. Why the bacillus should pass the lungs and become active in the glands has not yet been satisfactorily explained. This condition is a comparatively common sequel of whooping cough and measles, and is of a very frequent occurrence in foundlings. Exceptionally the tumors may attain a very large size.

The great danger of general infection from this mediastinal group has been referred to when considering acute miliary tuberculosis. It is claimed that the lung may be secondarily infected from this glandular focus. Even the pericardium may thus become involved. In many instances, however, it is impossible to say whether the primary lesion is in the glands or in the viscera.

**THE MESENTERIC FORM;** also known as *tabes mesenterica*, abdominal scrofula, and "consumption" of the bowels. In this form the disease involves the mesenteric and retro-peritoneal glands. The changes are similar to those occurring in other glandular groups, as already described. In this variety, if the abdomen is not too much distended by the intestinal contents, the diseased glands may often be discovered by examination through the abdominal parietes as individual growths or agglomerated masses. The condition may be a primary one, or it may result from tuberculous lesions elsewhere, as in the intestines or even in more distant parts. The primary form occurs mostly in children and in them



is not very uncommon. In adults, tuberculous enlargement of the abdominal, mesenteric or retro-peritoneal glands is always secondary to tubercular disease in other parts, particularly in the lungs. The attending symptoms are those of disordered digestive function, or of inflammation of the peritoneum. In the cases of children there are diarrhœa, offensive stools, canine hunger, emaciation, etc. In many cases most of the abdominal organs become involved, all being matted together by inflammatory changes. With the advance of the disease, the tuberculosis may become general, involving lungs, meninges, etc.

**Treatment.**—The above considerations concerning the pathology and clinical history of scrofula furnish very important indications for hygienic treatment. Fresh air, fresh and easily digestible food, proper clothing, and plenty of sunlight are strongly indicated, and are necessary factors in effecting a cure. In some cases, residence at the seashore for several months is invaluable. Mountain or country air is all sufficient in some cases.

Cod-liver oil is a good remedy in emaciated patients. In the stout and phlegmatic cases, it is not indicated. It may be given by the mouth or by inunction, according to the age of the patient.

Among remedies, the preparations of *iodine* and *lime* stand at the head of the list and should be used according to indications. *Hepar*, *mercurius*, and *silicea* are indicated in suppurative cases. The associated catarrhs and cutaneous eruptions often afford indications for *graphites*, *sulphur*, *lycopodium*, etc.

In obstinate cases, it is necessary to resort to surgical measures, *i. e.*, the removal of the diseased glands, lest general tubercular infection ensue.

## PULMONARY TUBERCULOSIS.

**Synonyms.**—Pulmonary phthisis; pulmonary consumption.

Tuberculosis of the lungs is the most frequent as well as the most interesting variety of tuberculous manifestation. On account of the destructive nature of the lesion, and the wasting and general impairment of nutrition, the word "phthisis" has long been applied. Phthisis is derived from *φθίειν*, meaning "to waste." The term consumption is most frequently applied by the laity.

So much has been written upon the subject of pulmonary phthisis, embodying such a variety in nomenclature in the application of the term tubercle, etc., that the student of this subject is liable to confusion. In order to "clear the way" for a definite understanding of the subject, we are probably justified in asserting dogmatically that the present state of our knowledge upon this subject warrants the statement that pulmonary phthisis presents a variety of lesions, the test of the character of which being not their minute anatomy, but the presence of the *bacillus*

*tuberculosis*. This being true, as we believe, it "brings order out of chaos." The lesions of phthisis embrace areas of consolidation of the pulmonary tissues of varying size, with varying degrees of degeneration, of excavation, and occasionally of healing. The consolidation is shown to be due to acute, subacute or chronic catarrhal pneumonic processes excited by the influence of the tubercle bacillus, to contracting interstitial pneumonia, and to the tubercle granulations. On the other hand, these inflammatory processes may be primarily simple and become infected later. But whether one or all of these pathological processes are present, or however one may predominate over the others, the specific bacillus is present in all. A simple catarrhal pneumonia, with its multiplication of alveolar epithelium and hyperplasia of the alveolar walls, or the constantly present bronchitis, is, therefore, as truly pulmonary tuberculosis as are the myriads of gray granulations which occasionally throng the pulmonary tissues. Clinical study of pulmonary phthisis leads us to the recognition of several varieties of that disease, these being based upon the character of the lesions, and largely upon the rapidity of their course. Embraced in this classification are cases of phthisis as rapid in development, progress and termination as a croupous pneumonia on the one hand, and those so protracted as to last for an almost indefinite period on the other.

In common with most recent writers upon this subject, I recognize: (1) Acute pneumonic phthisis, a tuberculo-pneumonic process running a rapid course, and presenting clinical features allying it in some instances to croupous pneumonia, and in others to broncho-pneumonia. Douglas Powell designates the former as the confluent form, and the latter as the disseminated form. (2) Chronic phthisis; (3) Fibroid phthisis.

The special pathological features of the great majority of cases of phthisis are: (1) Consolidation of the lungs of variable extent; (2) destructive changes occurring in the consolidated districts; (3) septic and tubercular infection.

CONSOLIDATIONS. Upon post-mortem examination, the lungs are found to be more or less extensively consolidated, this process being somewhat unusual at the base. One lung is, as a rule, more involved than the other, and in advanced cases of phthisis the signs of disease are in most cases much more marked in one apex than in the other. The region of greatest freedom is the base of the lung on the side opposite to the most diseased apex. Primary disease of the base is exceedingly rare, different observers stating the numerical relation of primary lesions of the base and apex to be from 1 in 200 to 1 in 500. Occasionally the apices may contain old, slight, latent lesions, and the base fresh developments. Such may easily be regarded as a primary lesion of the lower lobes by a careless observer. It is rare for one lung to be diseased, and

the other entirely normal. The consolidated areas are irregular in size and contour, and range in color from a whitish or grayish to a yellowish or greenish tint. The morbid changes begin in the apex of the organ and progression is towards the base, the oldest lesions being therefore in the apex. The order of invasion of the lung is quite regular, and has been carefully studied by Dr. Kingston Fowler, according to whom the earliest points of solidification appear in the upper lobe, but not in the apex, rather an inch or two below the latter, and usually external as well as posterior to the central point at this level. Extension from this focus is downward, and is probably largely due to the inspiration of the specific irritant. According to the direction of growth, the consolidation is detected more readily anteriorly, below the centre of the clavicle, and posteriorly in the supra-spinous space. Less frequently the primary lesion is detected below the outer third of the clavicle. General involvement of the upper lobe leads to gradual increase in area of the dulness and more marked auscultatory signs found in this region. The tendency to first attack the apex is manifest also in disease of the lower lobes, which, it will be remembered, extend posteriorly up to the spine of the scapula. The primary lesion here, according to Fowler, as in the upper lobe, develops one to one and a half inches below the apex, and a similar distance from the posterior border of the lung, and extends backward towards the posterior border of the lung, and laterally along the inner edge of the scapula, if the hand clasp the opposite shoulder, and the elbow is raised to the plane occupied by the shoulder.

*Nature of the consolidation.* Intra-alveolar accumulation of inflammatory products is the principal cause of consolidation of the lungs in pulmonary phthisis. Microscopical investigation of the consolidated areas demonstrates a variety in the character of the pathological changes. The processes involved, however, are all inflammatory in nature, and variously combined. They may be enumerated as (1) catarrhal pneumonic; (2) croupous pneumonic; (3) interstitial pneumonic; (4) miliary tubercle.

CATARRHAL PNEUMONIA, or, more properly speaking, a catarrhal broncho-pneumonia, is the most constant pathological feature of ordinary chronic phthisis. The terminal bronchial tubes appear to be the site of the beginning inflammation. The associated alveoli become involved, and are soon stuffed with inflammatory products, the predominating element being large, flat, nucleated epithelial cells, which are the product of the proliferation of the epithelial scales lining the alveoli. Upon section of a patch of broncho-pneumonic inflammation, an outer zone in which the alveoli are more or less filled with epithelial cells, which have undergone but little degeneration, may be detected. Internal to this is an area in which the alveoli are stuffed with epithelium which has undergone granular and fatty changes, and in some degree destruc-



tion, leaving a granular detritus. In a third, and the most central portion, the inflammatory products present a still higher degree of degeneration, in which the alveolar walls share. When degeneration is complete, this central region is reduced to a granular, structureless, caseous mass, in which the outline of the alveolar walls can be but faintly discerned here and there. As patches of broncho-pneumonia continue developing in the region of the primary focus, the gradual extension in size of each leads to fusion and solidification of large areas, even the greater part of a lobe. Occasionally quite extensive tracts are involved by simple peripheral extension of the primary focus. As there are usually many foci of inflammation in a well-developed pulmonary phthisis, and much disparity in their age, quite a variety of degrees of degeneration are met in the various consolidations.

In the more acute forms of phthisis we often discover a condition of the lungs approximating to that found in croupous pneumonia. Rapid progress, the presence of fibrin sometimes in considerable quantity in the alveoli, and the tendency to involvement of the entire lobe or even the whole lung, are the most prominent characteristics of this variety.

The amount of fibrin present in the alveoli is much less, however, than in croupous pneumonia, and the epithelium and leucocytes much greater, thus occupying a position intermediate between the catarrhal and croupous forms. Some authors apply the term caseous pneumonia, or galloping consumption, to this form. My own observations have led me to the belief that between typical forms of catarrhal and croupous pneumonic changes, due to the action of the bacillus tuberculosis, there is a great variety of combinations of these pathological conditions. In the typical broncho-pneumonic patch one occasionally finds a small area of more active change in which the alveoli contain some fibrin, and likewise in the croupous pneumonic patch, which has not been too rapidly developed, some areas manifest little but alveoli stuffed with epithelium.

**INTERSTITIAL PNEUMONIC CHANGES.** These are usually the more pronounced the older the lesion, and are consequently more pronounced in association with the catarrhal pneumonic patches, which often become encapsuled by a limiting membrane of fibrous tissue. The enclosed caseous matter becomes dry and infiltrated with lime salts. By means of subsequent ulcerative changes these "lung stones" may be freed and expectorated. If caseous degeneration of the patch has not occurred, a complete fibroid transformation of the pneumonic patch may take place, representing the most favorable result which can be expected.

**MILIARY TUBERCLES.** The nature and structure of the miliary tubercles have already been described. In the lungs they are typical. They spring most frequently from the alveolar walls, from the peri-vascu-

lar or peri-bronchial or the sub-pleural connective tissue. Whether developing in the alveolar or bronchial walls, they encroach upon their lumen and narrow it. The claim that such a growth of miliary granulation always precedes the pneumonic process, we think is questionable. They certainly are sometimes found absent upon most careful examination. The same can be stated regarding their secondary development about the consolidated areas, *i. e.*, they may or may not be present. If present, they are found in groups about the primary lesions, or widely disseminated in the lungs. The number of tubercles is usually greater when destructive changes have taken place in the lung. It is not uncommon to discover the lower lobes thickly studded in association with destructive changes at both apices. These granulations may be sclerotic, and this and other evidences of age suggest local chronic miliary tuberculosis.

CHANGES OCCURRING IN THE CONSOLIDATED AREAS, *viz.*, CASEOUS DEGENERATION, SOFTENING, ULCERATION, EXCAVATION AND SCLEROSIS. Concerning the occurrence of caseous metamorphosis in tubercular tissue, little need be added to what has been already stated. It is essentially a coagulation necrosis, and in its development bears some relationship to the acuteness of the phthisis and the extent of the lesion. The resulting softening, ulceration and excavation, lead to the formation of cavities, or "vomicae." These are usually found in the upper lobes near the apex, and vary in size from a barely appreciable excavation, to one the size of the fist. Occasionally much greater excavations take place, involving most or all of the upper lobe; and rarely, nearly the entire lung is found to have been destroyed. Considering the rarity of extensive cavities, physicians should discourage the exaggerated statements we so frequently hear to the effect that the "lung is gone," etc. Often a number of irregular cavities are found communicating with each other. In this manner, the lung may be "worm-eaten," like a locust tree, from apex to base. The interior of cavities may be smooth, with a well-formed limiting membrane (usually old cavities), or the walls may be rough, shaggy, and without a distinct development of fibrous encapsulating tissue. The formation of a limiting wall does not indicate a positive arrest in the progress of the excavation, many such continuing to enlarge by gradual involvement and disintegration of the adjacent lung-tissue. The most rapidly developing cavities and those attaining the largest size are the result of gangrenous destruction. The discharge from this variety is excessively offensive.

If cavities communicate freely with bronchial tubes, and are so related to them as to drain easily, they are readily evacuated by coughing efforts, and are therefore at times quite empty. As the vascular tissue resists the degenerative process longest, bloodvessels are frequently observed freely exposed within a cavity. Gradual weakening of their

walls and removal of the support furnished by the tissues, results in aneurismal dilatations, which frequently burst and cause hæmorrhage; such hæmorrhages usually occurring late in the disease and for apparent reasons being copious. The degree of vascularity of the walls varies greatly.

The slower the progress of the phthisis the greater is the development of fibrous tissue, which is nature's advance guard, established to arrest the progress of the enemy. If the tuberculo-pneumonic process has not advanced to the stage of softening, under favorable conditions, the affected area may undergo a sclerosis; the fibrous tissue developing into an encapsulating membrane only, or being distributed evenly through the diseased focus. An excavation sometimes contains bands of fibrous tissue extending across the cavity, and which may be mistaken for bloodvessels. If a lung consolidation has existed for some time, it is not unusual to find some degree of interstitial pneumonia in the portions of the organ which still appear free from tuberculous disease. This is indicated by an increased firmness, and often by visible development of connective tissue bands.

**CHANGES IN THE PLEURA.** The pleura is constantly involved in well-developed phthisis, but does not always contain tubercles. When present, they are visible as grayish-white minute bodies, or as caseous masses of larger size. The membrane is much thickened by repeated attacks of inflammation, especially that portion covering the apex. Adhesions may be slight or firm, and are often extensive. Effusion of a liquid exudate is infrequent. If present, it may be simple, or of a purulent or hæmorrhagic character. Pneumothorax is not common.

**CHANGES IN THE BRONCHIAL TUBES.** The larger bronchi are quite constantly in a state of chronic catarrhal inflammation. This process may extend downward, but probably much oftener, the minute bronchi are first involved, with extension upward and to the alveoli. The minute delicate bronchial tubes are apt to give way, as the result of the degeneration of their walls and the pressure from within, and thus develop bronchiectasic cavities. Involvement of the bronchial tubes occurs constantly in chronic phthisis. The mucous membrane is rendered uneven and the deeper tissues may be attacked. The peri-bronchial connective tissue is inflamed and thickened.

**CHANGES IN THE BRONCHIAL GLANDS.** The bronchial glands are usually enlarged and pigmented. In acute phthisis, miliary granulations, hyperæmia and œdema are usual. In protracted phthisis, caseous or cutaneous masses or purulent foci may be present. The danger of constitutional infection from this source has been referred to.

**DISTANT LESIONS.** In most cases of phthisis, especially if of long duration, lesions are discoverable in organs not immediately associated with the lungs; most frequent and important of these is tuberculosis of



the various organs. General tuberculosis and certain local developments have been referred to. It remains to mention laryngeal and intestinal tuberculosis, amyloid disease of the liver, kidneys, spleen, intestines and other organs. The endocardium has been shown to be the seat of tuberculosis much more frequently than was supposed, Kidd finding it in 5 per cent. of a series of five hundred cases.

**Clinical Course.**—The clinical course of pulmonary phthisis presents great variety in invasion, progress, and termination. So diverse may cases be, that it is only by the exercise of care in diagnosis that one can determine that all belong to the same affection. In support of this statement, it is only necessary to compare typical acute pneumonic phthisis and chronic phthisis in any of its forms. At no period of the disease is the above-mentioned difference in symptomatology as pronounced as in the incipency or invasion period. The symptoms of typical cases of phthisis, viz., insidious development with cough, dyspnoea, expectoration, hæmoptysis, fever, sweats, wasting, etc., are so firmly fixed in the professional mind, that there is danger of our overlooking cases of atypical beginning.

Unfortunately, this class is much larger than has been supposed. Confining observations to the period of development of the disease, we may easily isolate several groups of cases. A common variety may be designated as the fever group. In these fever is the predominating symptom. It may be intermitting, remitting, or continuous. Chilliness or severe rigors may attend, especially the intermittent form, and lead to a diagnosis of malarial fever. This error is very common, altogether too common. Attending this fever there is gradual failure in strength and flesh, and a development of anæmia, slight cough, and perhaps loss of appetite. Sweats may or may not be present. Later, symptoms forcibly directing attention to the lungs, such as cough, dyspnoea, expectoration, hæmoptysis, pain in the chest, etc., lead to a proper appreciation of the character of the disease.

In another group of cases, hæmoptysis is the first symptom to excite apprehension. The relation of hæmoptysis to phthisis is a most interesting question, and one which it will probably be impossible to settle positively. As physical diagnosis will not enable us to detect small lesions with certainty, especially if surrounded by healthy lung-tissue, we cannot determine in a given case whether the hæmorrhage precedes and is the exciting cause of the lung lesions, or whether an undiscovered lesion gives rise to the hæmorrhage. A case suggestive of the former cause came to my clinic at the Hahnemann Hospital. The subject was an iron-worker, thirty-six years of age, a large well-developed man with an excellent personal and family history. My first examination was within a few hours after the first hæmorrhage, and the most minute investigation failed to elicit anything suggestive of previous pulmonary

disease. The heart and other important organs were normal. After examination, I felt that it would be difficult to select a better specimen of healthy manhood, and yet this man while pursuing his usual work, and without evidence of unusual strain, suddenly expectorated a few ounces of clear blood. There were three recurrences of the hæmorrhage within a month. By the end of this time consolidation could be detected in the right apex. Even in such a case, however, one cannot say that the phthisis originated in the hæmorrhage. There is undoubtedly a want of sufficient evidence of the origin of phthisis in this manner, but it must be confessed that, even if it be true, it would be difficult to furnish testimony corroborative of the fact. Phthisis with hæmorrhagic onset is perhaps the least likely of any of the varieties to be misunderstood. In phthisis developing early, and with pronounced bronchitis, the question as to whether the lungs are, or are not involved, will usually receive attention. It is when bronchial or laryngeal symptoms of tuberculous origin develop more or less suddenly in persons supposed to be healthy, that the phthisical nature of the attack is not suspected.

The same may be said of pleurisy, which is occasionally a prominent early symptom. Frequency of the tubercular origin of pleurisy has of late been strongly advocated, some teachers contending that most pleurisies are of this nature. It has been shown that at least one-third of those recovering from acute pleurisy ultimately develop pulmonary phthisis. The dry localized form is the more frequent, although acute general pleurisy with effusion is occasionally met. In some quiescent cases large accumulations, due to inflammatory change, may take place without any acute symptoms.

The dyspeptic group, I have not found as prominent as the teaching of some authors would suggest. Feeble digestion is of course quite a common feature of phthisis in all its stages, but there does not seem to be a special perversion of the digestive function, which is suggestive of phthisis. It is rather the association of digestive disturbances with other symptoms, such as loss of flesh, anæmia, shortness of breath, etc., which leads to a suspicion as to the nature of the affection.

The group simulating pneumonia may be very deceptive for a time; indeed an acute pneumonic phthisis has not yet been generally accepted by the profession. These cases, of which I have met several, cannot be separated symptomatically from croupous pneumonia, which for a time they simulate closely. The fever in the tuberculous affection is said to present more marked daily remissions, but remissions of one to two degrees are surely not rare in croupous pneumonia. Auscultatory differences have been insisted upon, such as suppression of the respiratory sounds in pneumonic phthisis (Traube). The same condition may, however, exist in croupous pneumonia, and I am unable to corroborate this observation as applied to the tuberculous affection. With full develop-

ment at least, the bacillus is detectible, and in suspicious cases must be searched for in the sputum, which, if found, conclusively settles the nature of the case.

With the establishment of the disease we still observe great differences in the condition of the patient in the several varieties of pulmonary tuberculosis. The one suffering from acute pneumonic phthisis, with the attendant high temperature, feeble, rapid pulse, dyspnoea, pain in the side, and other symptoms common to acute pneumonia of the croupous variety, presents a picture differing much from that of the sufferer with chronic destructive phthisis with its extensive consolidation, large cavities, extreme emaciation, etc., or from that of cases of the same variety in which partial recovery has occurred with greatly improved general condition, but the patient walking about with, often, a cavity of large size; or that of the consumptive with fibroid phthisis, whose thin form, slight chronic cough, shortness of breath, and occasional attacks of bronchitis with troublesome cough and expectoration, lead us to look for the retracted chest-wall and dense lung-tissue. Again, any of the varieties enumerated may be extensively modified by the occurrence of one or more of the numerous complications to which they are subject.

The fatal termination of phthisis occurs in different ways, and at very different periods of the disease. In acute pneumonic phthisis death may take place within two or three weeks with much the same general symptoms present in croupous pneumonia; or the patient may pass into a typhoid condition; or, after rapid progress for a few weeks, the acute symptoms gradually subside, and a chronic state is established. In chronic phthisis death usually occurs from asthenia after several years of suffering, marked by alternate periods of arrest and progress. But life may be cut short at any time by a copious hæmorrhage, or repeated hæmorrhages, or rapid failure from excessive sweatings, high temperature, diarrhoea, general pleurisy, tubercular meningitis, general miliary tuberculosis, amyloid or fatty degeneration, etc.

The duration of the disease is subject also to the same wide variations. The most acute form may reach a fatal termination in the second or third week, and before softening has taken place. Others, less aggressive, last two to six months. A larger number prove fatal within two to three years, while others are held in arrest by treatment and climatic changes, and live from five to twenty or more years. In the case of long duration, death often results from complications, such as tubercular meningitis, intestinal tuberculosis, or amyloid degeneration; or, locally, from pneumothorax, empyema, pulmonary hæmorrhage, or severe bronchitis; while in the rapid forms the extent of lung-tissue involved is a most important factor. The progress of chronic phthisis is usually uneven. *It is by waves.* The periodical aggravations are apt to be ascribed to cold, which is undoubtedly the exciting cause in many instances. The



symptoms increase to an acme, then recede, and are followed by a period of better health. The remissions may be short or indefinitely protracted by good treatment, and the employment of climatic advantages. A few cases, after varying periods of activity, become latent, when practically good health is restored in some instances. This statement is justified by finding in the apices of the lungs of such persons, who have died of other affections, cicatricial and pigmented patches, including perhaps cretaceous formations.

**Symptoms.**—In view of the variety which exists in the lesions of phthisis, its varying duration, the great number of complications, which may occur, and the number of organs which may be involved, it is difficult to arrange the symptoms in an orderly manner. It is perhaps most convenient to consider them in two groups: (1) local symptoms; and (2) general symptoms.

**LOCAL SYMPTOMS. CHEST PAINS.** Pain may be a prominent symptom in some cases, while in others it is practically absent throughout the entire course of the disease. Some observers have given special diagnostic importance to certain pains, such as cutting pains between the scapulæ; their significance is, however, doubtful. Acute pain is most frequently due to pleurisy, and may be referred to any portion of the chest. Patients are prone to locate their lesion by the situation of the pain. When it is due to pleurisy, it possesses some reliability as a guide to the seat of most extensive lesion. The frequency of the association of myalgia and intercostal neuralgia with phthisis has not been made sufficiently prominent, many such cases being looked upon as of pleuritic origin. The strain of coughing may cause soreness of the chest muscles, pain at the insertion of the diaphragm, and in the abdominal muscles.

**COUGH AND EXPECTORATION.** Cough is the most notable phenomenon of the disease, because it is usually the first symptom, and an almost invariable attendant. The laity understand this, and patients with incipient phthisis are often unwilling to acknowledge a cough. For this very reason care must be taken not to be deceived by the absence or mildness of the cough. Cases may also arise in which cough is very slight, even with the presence of cavities. As every variety of cough may be present in phthisis, and as there is nothing pathognomonic in the phthisical cough, it is unnecessary to describe it, further than to say that it is usually dry in the early stages; later it becomes moist, paroxysmal, and is often aggravated at night. Cough, if excessive, produces exhaustion and frequently sleeplessness, as well as failure in nutrition from vomiting of food. Like cough, *expectoration* is quite constantly present. It may also like cough be absent, even in advanced disease. At present I have a case of rather rapid phthisis with quite extensive consolidation, and a moderate cough, but with so little sputum that the

patient has not been able to furnish me sufficient for microscopical examination. The sputum is at first catarrhal, but with the progress of the disease it becomes free, muco-purulent, and may be very profuse. With the appearance of excavations in the lung, the sputum develops the peculiar features which have led to the term "nummular" or money-like sputum. At the same time, expectoration is apt to be periodical, occurring most copiously in the morning, at which time the cavities are full from the night's accumulation. The sputum now consists of a heavy grayish or greenish-yellow muco-purulent matter, which possesses sufficient consistency to allow of its being rolled into a mass, in the cavity or bronchial tubes, during the efforts at expectoration. These masses contain small, round, yellowish opaque bodies. Such sputum resting in a thin fluid is quite suggestive of phthisis. It is an error, however, to suppose that it depends, always, upon a cavity for the development of its peculiarities, for it may be present when cavities do not exist, as I have been able to determine post-mortem. During recent years the sputum has acquired a new importance, for the reason that it has been discovered to contain elements of an invaluable character in making a diagnosis, viz., the bacillus tuberculosis and yellow elastic fibres. The former is proof positive, the latter strongly suggestive of the existence of pulmonary phthisis. To discover the elastic fibres, boil a quantity of sputum in a solution of caustic soda for fifteen or twenty minutes, or until liquefaction of the mass has taken place. If the solution is now allowed to settle in a conical glass for a few hours, the elastic fibres will settle, and may be removed by means of a pipette for examination with the microscope. For this step all that is required is to cover a drop upon a slide, and examine with a low power. These fibres may be sometimes seen in thin layers of sputum with the unaided eye. The presence of elastic fibres in the sputum indicates the presence of a destructive lesion, and the destructive lesion of the lungs which greatly predominates over all others is phthisis. By the appearance of the fibres one can state their origin with considerable certainty. Fibres from the alveolar walls are branching and often map out the shape of the alveolus from which they are derived. The elastic fibres of the bronchi may be found in an elongated reticulum. These fibres are long and several may be in close juxtaposition. Portions of small bloodvessels are occasionally present, also alveolar epithelium. Concerning the latter, it is said that alveolar epithelial cells do not appear in the sputum of ordinary bronchitis before the age of thirty years. It is not rare in cases of phthisis with cavities to have at intervals sputum thoroughly intermixed with blood, the latter originating in capillary rupture. The color may be bright red or brownish. The pneumonic process, which is frequently set up about tuberculous foci, may be attended by the characteristic "rust-colored" sputum of croupous pneumonia. Fœtid sputum indicates that a gangrenous process has developed, or that bronchiectasis

coexists. Various bacteria and a variety of fungi are present, especially if there are cavities of size. The method of examination of the bacillus has been given in another section. The number of the bacilli present in the sputum varies much in different cases, and fluctuates in the same case. There appears to be a relationship existing between their numbers and the activity of the disease, a relatively large number suggesting a well-developed and active lesion. In cases of phthisis with expectoration the bacilli may be found quite early and are then of invaluable diagnostic importance. Examinations for this organism require a good one-sixth inch objective, but a one-tenth to one-fifteenth immersion lens is much more satisfactory. Blood is not frequently present in the sputum. Its source may be (1) hyperæmic tracts of bronchial or lung tissue; (2) eroded vessels. In the early stages of phthisis, and later, during intercurrent attacks of acute or subacute bronchitis, the sputum may be tinged with blood or there may be streaks or small clots of blood which are due to hyperæmia. The free expectoration of blood, however, which occurs later, is due to erosion of bloodvessels, and may vary from a few mouthfuls to hæmorrhage proving fatal. Copious hæmorrhages occur most frequently from rupture of aneurismal vessels which are exposed in cavities. Were it not for the thrombi which form in the exposed vessels of the highly vascular lung-tissue, hæmorrhage would be a much more frequent symptom of phthisis. Its diagnostic significance is well understood by the laity, patients expectorating blood being usually fearful of its portent, attempt to explain it away. The relative frequency of hæmoptysis in different cases of pulmonary phthisis varies greatly. I have repeatedly met cases with physical changes which could be detected only by the greatest care in examination, yet who suffered from repeated bleeding, and at intervals for years. In one case of this character in which I have had special opportunity to follow the lesion, there was little increase for three or four years; then arrest, and the patient has now been practically well for several years. In others with the most extensive lesions, even numerous excavations occur without hæmorrhage. When blood is expectorated in quantity, it is bright red, frothy, and more or less coagulated. When bleeding occurs slowly, the blood lies in the respiratory passages or in cavities, and becomes dark or well coagulated before expulsion. Slight bleedings do little harm, and occasionally the patient feels relieved. But in spite of this it is better to treat blood-spitting, even if slight, as an important symptom, as it is so often the forerunner of freer discharges. Free hæmorrhage not only weakens the patient, but often exercises a serious influence upon the course of the disease, being often followed by quite rapid and extensive consolidation of the affected lung. This is mainly of a pneumonic character, and in case of subsequent improvement, much of the consolidated lung-tissue may clear up.

**DYSPNŒA.** Most phthisical patients have an increase in the number



of their respirations, and while these are still further increased by exertion, there is seldom a complaint of dyspnœa, even though much of the lung-tissue is incompetent. This is partly explained by the anæmia, the reduction of the corpuscular elements of the blood leading to a lessened demand for oxygen. The great reduction in bulk of tissue, which takes place during the wasting of phthisis, also leads to less demand upon the blood. Decided dyspnœa is the result, especially of pleuritic pains, adhesions, rapid and extensive consolidation of lung-tissue, miliary tuberculosis, or large effusions into the pleural sacs.

**Physical Examination.** — Physical examination of phthisical patients makes known (1) in certain cases, abnormalities in the physical character of the chest, as well as certain general conditions which collectively constitute the strumous or phthisical habit; and (2) the physical signs indicative of consolidation, softening, or of cavities, all of which may exist in the lungs at the same time.

On **INSPECTION**, in a certain percentage of patients with phthisis, the thorax presents characteristics which have led to the appellation of “paralytic” or “phthisical” thorax. The most important features of such a chest are unusual length, associated with smallness, flatness and abnormal width of the intercostal spaces. The scapulæ project like wings, and the epigastric angle is abnormally acute. With advancing disease the chest is flattened at some point or points, especially over one or the other apex, this being especially noticeable anteriorly in the supra- and infra-clavicular regions. The respiratory movements are diminished in all stages, and this diminution is usually more pronounced over the apex region of one lung, although it may rarely be more noticeable at the base. Impaired expansion is usually rated as an early sign of great importance, but if well-marked, a considerable degree of structural change has probably already taken place. If this is true the sign cannot be considered as relating so especially to the earliest period of the disease. The supra- and infra-clavicular regions, as will be suspected, most frequently manifest a defective expansion. The condition of the expansion is usually best observed by standing behind the patient and looking downward upon the anterior surface of the chest. Abnormal diffusion of the cardiac impulse is often observed in disease of the left upper lobe. It may be observed as high as the second interspace. Pleuritic adhesions and thickening, and especially fibroid phthisis, lead to well-marked contraction and flattening of the chest, with defective expansion upon inspiration.

**PALPATION.** Defective expansion diagnosticated by inspection may be corroborated by palpation. Vocal fremitus may be increased over the same area, and is detected by the touch (tactile fremitus). Most careful attention should be given to the regions above and below the clavicle, which correspond to the suspected apex. Flint particularly

called attention to the greater degree of fremitus at the right apex of the normal chest. Increase of tactile fremitus is also common over cavities.

**PERCUSSION.** Pulmonary resonance is affected by the various phthisical processes, which cause varying degrees of consolidation or destruction with excavation of lung-tissue. The degree of dullness depends upon the extent and density of the consolidated area, remembering that a lesion situated near the surface of the lung, close beneath the chest-wall, which would give rise to well-marked impairment of the percussion note, will, if more deeply situated, and surrounded by normal or emphysematous lung-tissue, impair resonance little or not at all. The higher pitch of the percussion note over the dull or diseased region was forcibly called to our attention by Flint, and will be found a most valuable discriminating factor. The locations in which altered percussion is most likely to be found are the clavicular regions, the supra-spinous spaces posteriorly, and the inter-scapular space, especially when the arms are crossed in front and the hands made to clasp the opposite shoulders. Percussion is usually carelessly performed, and is consequently of little value to such observers in the early stage. The necessity of comparing the diseased side with the sound or less impaired one, must never be forgotten. Cavities with thin walls yield the "cracked-pot" sound. The highest degree of loss of resonance occurs in old cases of fibrous phthisis. Rapid changes in the percussion note are often due to pneumonic consolidation about tuberculous foci, or equally rapid absorption of the same.

**AUSCULTATION.** The auscultatory signs of value may be enumerated as: (1) Feebleness of the respiratory sounds; (2) prolonged expiration; (3) harshness or roughness of respiration; (4) cogged-wheel breathing; (5) râles; (6) signs associated with cavities; (7) signs related to the pleura.

*Feebleness of Respiration.* Persistent diminution of the respiratory murmur at one apex is common in early phthisis, and is often combined with impaired expansion of the chest-wall over the same area. Care must be taken to distinguish between such a localized impairment and a general feebleness of the respiratory sounds, the latter being quite common in persons who are illy-developed muscularly, who breathe imperfectly, or occasionally in those who may be temporarily debilitated.

*Prolongation of expiration* is one of the most important signs of a commencing pulmonary consolidation. If found at the right apex, however, there must be an association with other modifications, in order to be significant of disease. When this sign stands quite alone, which is rarely the case, its pathological character may be sometimes determined by the gradual extension of the area over which the sign is heard. When pathological, the expiratory sound is also, as a rule, dis-

tinctly higher in pitch than the inspiratory sound, and is also usually separated from it by an interval. The associated inspiration is generally higher in pitch, and harsher in quality, than in the same region upon the opposite side of the chest. These several modifications of the respiratory sounds, variously developed and combined, constitute the so-called "harsh" or "rough" respiration, or, more properly, broncho-vesicular respiration, which is the most important of the early signs of pulmonary phthisis. It is often necessary, in feeble persons, to voluntarily increase the force of the respirations, in order to develop the presence of abnormalities.

**COGGED-WHEEL RESPIRATION.** A localized interruption of the respiratory sound, which oftener occurs in association with inspiration, is frequently present in phthisis, but, standing alone, possesses little diagnostic value, as it may be due to other causes.

**LOCALIZED BRONCHIAL RÂLES** are strong corroborative evidence of phthisis. They may be dry or moist, and indicate a catarrh of the fine bronchial tubes involved in the diseased area of lung. Pneumonic processes set up about primary lesions may be indicated by crepitant râles. Quite as important as a corroborative sign is the presence of a localized pleuritic friction sound.

**SIGNS OF CAVITIES.** In advanced phthisis we often find superadded to the signs of consolidation, those indicating cavities. The localization of a pulmonary cavity by means of physical signs is often easy, but perhaps just as often requires repeated and painstaking observations. Many cavities are never detected during life. A cavity may be suspected if over a limited area there is persistent bronchial breathing, perhaps somewhat amphoric in character, combined with but little or no dulness upon percussion. The character of the percussion note over cavities depends upon the thickness of consolidated or air-permeated tissue overlying the excavation, so that the percussion note may vary from marked dulness to a tympanitic character. Tympanitic resonance within a limited space bounded often by a larger area of percussion dulness is perhaps most important. The cracked-pot sound is present when cavities are superficial and communicate freely with a bronchus. In well-developed cavities, there may be heard gurgling râles, and amphoric or cavernous breathing, vocal resonance may be increased (bronchophony), or there is perhaps distinct pectoriloquy. Wintrich has called attention to the increase of the tympanitic character of the percussion note, when the mouth is opened widely. Gerhardt has observed also another feature of cavities, *i. e.*, that the pitch of the percussion note often changes with a change of position of the patient.

**PHYSICAL SIGNS IN RELATION TO THE VARIOUS STAGES OF PHTHISIS.** The frequent practice of treating the subject of physical diagnosis in relation to the several stages of phthisis has been avoided in this article, as all the stages may be present in one lung at the same time.



**General Symptoms.**—**FEVER** is the most important of the general symptoms of phthisis. Every type of fever is represented, *i. e.*, intermitting, remitting, and continued. An afternoon and evening rise of temperature is the type most frequently observed. This is often associated with a morning subnormal temperature. The extent of the daily fluctuations is a very good measure of the activity of the case. Chills or chilliness may initiate the fever paroxysms in any stage of the disease, but especially after destructive changes have taken place (old phthisis); pronounced rigors are not common, when the fever type is pyæmic in character. The frequency with which phthisis is attended by an intermitting form of fever in its early stage, has led to many errors in diagnosis. In Philadelphia one is often called to a case of “malaria,” which physical diagnosis demonstrates to be pulmonary phthisis. Too many of these errors are committed by the profession.

**NIGHT SWEATS** occur at some period in the course of nearly all cases. They are most frequently encountered towards morning, although they may occur earlier in the night, and even during the day, while sleeping (sleep-sweats). They are occasionally present in a very early stage of phthisis. Rarely, the sweating is almost continuous. I have observed this in rapid pneumonic phthisis. So great is the loss under these circumstances, that the patient may present collapsic symptoms. So important is fever as a symptom in early phthisis, that the detection of a daily afternoon or evening rise, without apparent cause, should excite at once a suspicion of incipient tuberculosis. Frequent observations should be made during the twenty-four hours in all cases with fever. In this way only can we hope to secure an accurate observation of the temperature range. The duration of the fever in the intermittent form, and this is the most commonly met with, is variable. In some the paroxysm is over in two or three hours. In others it is continuous from eight to twelve or fifteen hours. Slight fevers the patient may not be conscious of; higher grades excite the general symptoms common to fever, which attract attention. A continued form of fever is of more serious import than that marked by intermissions. It is frequently symptomatic of a complicating pneumonia.

**PULSE.** The pulse is generally decidedly feeble and readily accelerated. Some look upon this excitable weak pulse of early phthisis as quite characteristic. It is seldom disturbed in its rhythm.

**EMACIATION.** Loss of flesh is a prominent symptom in all cases of chronic phthisis. One seldom sees greater emaciation than is present late in the course of the disease. In most cases there is quite a close relationship between the body weight and the progress of the lesion. An arrest of the progressive loss, or a slight gain, is always a favorable indication. Loss of flesh frequently occurs early, even before the nature of the affection is settled, and is then of diagnostic value. The blood suffers

deterioration also, anæmia being indicated by the pallor, which is present in all cases. The ænæmic state may develop early. In a case recently under my care, a diagnosis of pernicious anæmia had been made. A very slight, hacking cough attracted attention and resulted in the discovery of a case of phthisis in which, for a time, the local symptoms were insignificant. The subsequent progress of the case corroborated the diagnosis. Anæmia is often concealed by the flushed lips and cheeks attendant upon the hectic fever.

**NERVOUS SYMPTOMS.** The mind usually remains perfectly clear, even through long months of fever, waste, and distressing local symptoms. Consumptives are proverbially hopeful, even late in the course of their disease.

**GASTRO-INTESTINAL SYMPTOMS.** Tuberculous ulcers are sometimes found on the soft palate, base of the tongue, tonsils, etc., and lead to considerable local disturbance. Much diagnostic significance has been attached to a "red line" on the gums, but it has been shown to occur in many other diseases. Snader's observations upon this symptom are valuable. Aphthous ulcers are common. Tuberculosis of the pharynx, and especially of the larynx, separately or associated, interfere with swallowing, and hence with nutrition. The appetite is variable, and often fails in the late stages. Nausea and vomiting are common symptoms, and may be due either to disturbances of the stomach itself, or to distant lesions acting through the vagus. Dyspepsia is common, but does not appear in a characteristic form, as has been taught by some. The especial loathing of fats, attributed to the dyspepsia of consumption, I have not been able to determine to be at all common.

**DIARRHŒA** may be present at any period of the disease, especially in the later stages. It is due to various causes. Tuberculous ulceration of the colon or ileum is the most frequent. A few are due to indigestion, catarrhal inflammation, or lardaceous disease. The various organs within the abdominal cavity may be attacked by the lardaceous or fatty change, and originate a variety of symptoms related to the kidneys, liver, etc. Perforation of tuberculous ulcers of the bowels or the development of tubercles in the peritoneum may give rise rarely to peritonitis.

**CIRCULATORY SYSTEM.** The heart is affected by endocarditis in fully 5 per cent. of all cases. Harsh apex murmurs not due to endocarditis, occur rarely. A pulmonary systolic murmur may appear at any period of the disease. The uncovering of the heart by the retracting upper lobe of the left lung has been referred to. Pulsations of the capillary vessels and of the veins upon the back of the hands have been noticed. Femoral thrombosis, especially upon the left side, is not infrequent in advanced phthisis. General miliary tuberculosis, tubercular meningitis, tuberculosis of joints and bones, Addison's disease, acute parenchymatous nephritis, etc., are occasional complications.

TUBERCULOSIS OF THE LARYNX is a frequent complication of chronic pulmonary phthisis. It may develop at any period, but usually after free development of the pathological process in the lungs. The tubercles consist of minute accumulations of cell-elements in the mucous or sub-mucous tissues, and are visible as slight prominences. The results are catarrh, œdema, ulceration, perichondritis, and necrosis of the laryngeal cartilages. The most frequent points of attack are the mucous and sub-mucous tissues covering the epiglottis, the aryepiglottic folds, the arytenoid cartilages, ventricular bands, and the vocal cords.

So constant is the association of pleurisy with phthisis, that it may well be considered a feature of the disease. The dry form is the more common. Lesions near the surface of the lung are usually covered by thick layers of the lymph. In old cases of phthisis, it is common to find the apex capped with thickened and united pleural layers. When accumulations of fluid occur, they are more apt to be purulent than simple.

PNEUMOTHORAX develops most frequently in association with phthisis, according to West, in 90 per cent. of all cases. Still, the percentage of cases of phthisis in which this complication occurs is small. Bursting of a cavity into the pleural sac is the common cause. Pleurisy with a serous or more often a purulent exudate develops, and thus a hydro-pneumothorax or a pyo-pneumothorax is superadded.

SKIN. Pityriasis versicolor occurs quite frequently, especially over the chest. The œdema of the feet and legs, which usually comes on late, is due to cardiac weakness or to femoral thrombosis. Lupus, which we now know to be a specific tubercular disease of the skin, is but rarely observed in connection with pulmonary phthisis.

**Fibroid Phthisis** is a condition of induration and contraction of the lung (tuberculous interstitial pneumonia), usually developing gradually upon an arrested typical chronic pulmonary phthisis, or supervening upon a chronic pneumonia or pleurisy. Fibroid changes also involve the pleura, and the bronchial tubes may become more or less dilated and otherwise altered. In some degree, fibroid phthisis is a common feature of chronic destructive phthisis. It frequently affects one lung only, and is in the type of a chronic affection. It is more common in the apex, where it often surrounds cavities, than in other portions of the lung. The special clinical feature is a shrinkage of the affected lobe and retraction of the overlying chest wall. The heart may be abnormally exposed by the contracted lung, and displaced to the affected side. The normal lung often presents an extension of its area of percussion resonance, due to emphysema. The liver or the spleen may be displaced upward. The existence of a unilateral non-tuberculous contraction of the lung is doubted.

The lung is often found shrunken to less than one-third its normal



size, and bound to the chest-wall by a dense fibrous stratum. The lung consists of tough grayish or whitish fibrous tissue, containing ulcerating cavities, dilated bronchial tubes, caseous matter, and cretaceous masses. The second lung is usually slightly affected at the apex.

The symptoms are cough, usually with purulent expectoration, due to the frequently associated bronchiectasis and dyspnœa, with little constitutional disturbance. Fever and sweats are slight or absent. The patient is thin and anæmic. Rarely profuse hæmorrhages may occur from aneurismal vessels exposed in a cavity. Aside from the displacements due to contraction of the sclerotic tissues, the various signs of consolidation and, perhaps, of cavities, are present over the affected area. Sequential to the chronic suppurative process, a more or less general amyloid degeneration may develop. The duration of fibroid phthisis may range from three to twenty or even thirty years.

The diagnosis must depend upon the discovery of the bacillus tuberculosis in a case presenting physical signs of consolidation and contraction in association with the history.

**Diagnosis.**—Since the discovery of the bacillus tuberculosis the problem of diagnosis, for the great majority of cases at least, has been greatly simplified. A little sputum on a slide is usually sufficient for a prompt and positive opinion. In most instances, however, physical examination of the chest, in association with the history and clinical features of the case, is sufficient for an immediate opinion. Early detection of the disease is of the greatest importance. The auscultatory signs are the most valuable at this early period; especially suggestive is the discovery at one or the other apex of some degree of broncho-vesicular breathing, and the râles of localized bronchitis. Whenever doubt exists it is wise to treat the case as one of phthisis. The most common errors are: (1) Mistaking the general symptoms of phthisis, in cases in which local symptoms are not prominent, for malaria and other affections of a general character; (2) in mistaking phthisis, with predominant local symptoms, for one of the various inflammatory pulmonary diseases. With reference to the first group, it is to be remembered that in well-settled regions of country malarial affections are not nearly so common as believed. Our most competent clinicians in the large eastern cities tell me that phthisical cases are very frequently reported as malarial, which statement agrees with my own experience. Ignorance of physical signs, carelessness, or rather neglect in physical examination, is responsible for the error.

When the onset of phthisis is rapid and simulates laryngo-tracheal or bronchial catarrh, or pneumonia in some form, the diagnosis is often difficult without the aid of microscopic examination of the sputum. The bacillus, as well as the elastic fibres, may be found in the sputum at a very early period of the disease, frequently before physical signs of con-

solidation are present. It seems necessary to recall the fact that in a very few cases of consumption the bacillus is not found. Its absence, therefore, does not prove that the patient is not phthisical. Some authors are inclined to admit an apex catarrh, which is not tuberculous. While it appears reasonable that such may be the case, a catarrh of this nature must be rare, and is exposed to the great risk of becoming infected and ultimately tuberculous.

Amann recently published the results of 4,000 examinations of sputum taken from 1,792 patients. Koch's bacillus was present in 83 per cent. of the individuals. Frequent examinations were required for its detection in about 30 per cent. In a group of 856 cases, which were under continued observation, there was a complete and permanent disappearance of the bacilli in 16 (1.7 per cent.); a marked diminution in 144 cases (16.8 per cent.); a marked increase in 254 cases (29.7 per cent.); in 442 cases (51.5 per cent.) there was no change. In 8 patients whose sputa were continuously free from bacilli, elastic fibres were found; 4 of these were bronchiectasis, 3 were of doubtful nature, 1 was pulmonary abscess. Elastic fibres were absent in 11 per cent. of those clearly tuberculous. In the remaining 1,331 patients both elastic tissue and bacilli were found, showing the bacilli to be more constantly present than the elastic fibres. According to Amann there is a positive parallelism existing between the number of bacilli and the progress of the disease. He further states that a cure of pulmonary tuberculosis cannot take place without a complete and permanent disappearance of the bacilli.

The employment of inoculation methods for diagnostic purposes has received considerable attention. Delépine has recently made twenty-eight experiments upon guinea-pigs with tuberculous matter from various sources and concludes that results may be easily reached in two or three weeks. Positive results he considers as giving more definite information than the discovery of the bacillus, and that the negative results are nearly as valuable as the positive.

The author desires to emphasize the importance of an early diagnosis and intelligent treatment of pulmonary phthisis. There is too much of a tendency to obscure the disease in the patient's eyes, during the early stage, which results partly from want of confidence in the success of treatment, and partly from a disposition to protect the patient from the possession of unpleasant knowledge. This course is usually followed by the worst of results, both for the patient and physician—the former frequently failing to continue treatment with that fidelity which would be exercised, did he know the serious nature of his disease; the latter in turn being accused of failure to comprehend the nature of the case in its early stage. It is a melancholy fact that scarcely one-third of the cases of pulmonary tuberculosis are positively diagnosticated by the medical attendant until the symptoms are recognizable by almost

any intelligent individual. Physicians who do approach these cases in the proper spirit, and make early diagnoses, are quite apt to be designated "alarmists." Not only is a proper diagnosis and treatment necessary at an early day, but the intelligent co-operation of the patient is essential to a favorable result. This can seldom be secured without a plain statement of the case. With the exercise of proper tact in its communication the patient is seldom alarmed by the information, or if somewhat excited, is quickly calmed. The treatment of phthisis requires great persistence, often after the symptoms have subsided, and the patient fancies he is well. Indeed, this is a danger period, unless the patient fully understands the nature of his disease. Another requisite to the successful treatment of phthisis is a physician in attendance who has confidence in his resources. Too many are imbued with the idea that treatment is attended by but temporary improvement, and there is a consequent half-heartedness in the zeal with which treatment is pursued.

**Prognosis.**—The subject of prognosis in pulmonary tuberculosis is a large one, Pollock having written a good-sized treatise upon it. Perfect recovery from pulmonary tuberculosis, which has been sufficiently well advanced to give rise to well-marked physical signs, never occurs. When a practical cure takes place, and this is not uncommon, the areas which have been tuberculous are represented by scars of fibrous tissue, which may be associated with calcareous deposits or cheesy foci. Pathologists have been surprised to discover the large number of persons dying from diseases other than tuberculous, whose lungs present the evidences of obsolete or inactive tuberculous lesions. The percentages reported vary from about 5 to 75 per cent. of autopsies. This is a wide discrepancy, but it is explainable. The majority of observers report percentages ranging from 25 to 40.

Even in cases of apparent cure, we are never certain of the complete extinction of the disease, and therefore that it may not at some future time again become active. In acute pneumonic phthisis the prognosis should always be unfavorable, although in a few cases its progress is arrested, and the disease becomes chronic. In the ordinary chronic destructive variety, an opinion is reached after consideration of the extent and stage of the lesion, as well as of the degree of constitutional disturbance. Laryngeal and gastro-intestinal complications are especially unfavorable. Tubercular meningitis, fatal hæmoptysis, or pneumothorax, may develop at any time. Unexpected progress and unexplainable arrests are not rare. The patient's ability to secure good food and suitable climatic change, as well as his faithfulness in carrying out the treatment, have a powerful influence upon the course of the disease. Of individual symptoms a continuous high temperature, rapid loss in flesh, and feeble digestive power, are especially unfavorable.

Strümpell thinks the temperature of great prognostic importance in



pulmonary tuberculosis, considering afebrile phthisis as of mild type. Subfebrile cases, in which there is slight rise in the evening (and often a subnormal temperature in the evening), are looked upon as less favorable; while the typical hectic fever is indicative of rapid progress. Strümpell considers that a remedy which does not control the fever is inefficient against the disease.

The average duration of the disease, according to the large statistics of Pollock, which are corroborated by other observers, is about two and one-half years. Statistics from private practice, which represent the course of the disease in the better classes, indicate the duration as more than double that stated by Pollock.

A person possessing the evidences of active tuberculosis of the lungs should not marry. Danger to offspring is probably greater from the mother.

**SPONTANEOUS RECOVERY.** That tuberculosis may terminate spontaneously in recovery is undoubted. This is shown, as already indicated by the frequency with which old scars are found in the lungs of persons dying of other diseases, by the frequency, too, with which we observe the disappearance of elastic tissue and the bacillus tuberculosis from the sputum of persons in whom lesions can be demonstrated, from the influence of climatic change alone. The scars referred to simply indicate areas of fibrous tissue which have undergone contraction, for without doubt cavities of any considerable size do not undergo cicatrization. Cavities undergo shrinkage in size and are surrounded by condensed tissue and continue to secrete a little purulent matter, or may occasionally become quite dry. The frequency with which spontaneous recoveries take place, may be judged by the statistics of Heitler, of Vienna, who found 780 instances of obsolete tuberculous lesions in a series of 16,562 post-mortem examinations. Other reliable pathologists have reported percentages as high as 39.

**Treatment.** — **PREVENTIVE.** The profession is awakening to the importance of the preventive treatment of tuberculosis. As the specific agent, the bacillus tuberculosis finds its way to the outer world almost entirely by means of the sputum, it is of the first importance that this dangerous agent should be promptly destroyed. Every consumptive should use a "spit-cup," which should always contain a solution of an efficient germicide. The filthy and dangerous habit of expectorating into the handkerchief, along the streets (and even upon the floors of the house, by the lower classes), cannot be too strongly condemned, as the dried and powdered sputum, becoming mingled with air-currents is liable to be inhaled. Consumptives should sleep alone, as the risk of contagion to those who happen to room with them is considerable. They should not marry, nor should a diseased mother suckle her young. After all possible care has been taken in the directions above indicated, one may still become

infected by food taken from tubercular animals, especially bovines. The milk of cows has been demonstrated to be more dangerous than beef. So great is this risk, that all herds of milch-cows should be regularly inspected by able and properly constituted authorities, and all tuberculous animals should be at once destroyed. Skilled inspection of cattle which are to be used as food, should also be made before the sale of their flesh is permitted. Such rigid measures can only be carried out generally and satisfactorily by the State. When an hereditary tendency can be determined to exist, precautionary measures should be adopted, even in early life. In the infant and young child care should be bestowed upon diet, bathing and clothing. All affections of the respiratory apparatus should be promptly treated and removed as soon as possible. Catarrhs of the bronchi should always be looked upon as a menace, and treated with the same care bestowed upon serious forms of disease. Chronic changes in the nose, naso-pharynx or tonsils are of especial importance. With the approach of adolescence, the danger is greatly increased. A nutritious diet, gentle but persistent exercise of various kinds, good part-wool underclothing worn during the entire year, plenty of sleep, avoidance of hard study; light, well-aired apartments, free from dust, must be insisted upon, if the patient's circumstances will permit. Rapid growth at this period must be looked upon with suspicion, and a double watch accordingly put upon the patient. As Fothergill aptly states it, rapid growth is suggestive of apical mischief. Of all measures, however, a change to a suitable climate is most beneficial.

CLIMATE. Concerning the best climate for phthisical patients, there are various opinions. Many patients improve for a time in almost any climate differing somewhat from that to which they have been accustomed. In Philadelphia, for instance, a change to the seashore, or to the mountains inland, is usually followed by a reduction in temperature, less cough, better sleep, and an improved general condition of health; and, if the case is not too far advanced, occasional changes to these points may, in association with good treatment, result favorably. But, for most persons, a longer residence in a more equable and dryer climate is required. Regions possessing proper qualifications are to be found in the western portion of the United States. Most beneficial are the great plains bordering on the Rocky Mountains. The advantages of this region are, first, that almost any desirable altitude can be secured, a proper degree of which stimulates respiratory exercise; secondly, it possesses a high degree of dryness, one of the most important factors, especially on account of its inhibiting the growth of the tubercle bacillus; thirdly, it presents the opportunity for a pleasant variety of life apart from towns and cities, *i.e.*, "ranch life," which is the ideal mode of living for the phthisical, if good food and a contented mind can be secured. For those who desire the advantages of a better-settled country, with all

this implies, Southern California is to be highly commended. In the East, the Adirondacks, Northern Maine, the mountains of Virginia, North Carolina and Georgia; and, especially in winter, the flatter country of Florida and Southern Georgia, are all valuable health resorts for this class of patients. In Europe, one may employ the tonic and stimulating high altitudes of Davos, St. Moritz, or Wiesen in Switzerland; or, the more southern health resorts. Of the latter, the best known are the islands of the Mediterranean, the Italian and French Riviera, especially Nice, Mentone, Cannes, and San Remo. The characteristic feature of these southern resorts is a warm climate, varying in the degree of heat, moisture, wind, etc. Some are marine towns, with a moist climate. *e. g.*, Madeira, and are sedative in their action; others are dry, marine resorts, possessing a more tonic influence. The selection of one of these points for the patient to reside in must be determined by the nature and demands of the case, and also by the season of the year. There is no doubt but that the tendency in professional opinion is towards the employment of a high, cold climate, if a change is resorted to in the earlier stages; but there is quite a percentage of the old, the weak, the nervous, the gouty or rheumatic, and those suffering from organic heart disease, etc., who are more comfortable, and improve more rapidly, in the lower and warmer regions. In considering the subject of climate for the phthisical, it will be observed that the regions in highest repute are those which are sparsely settled, and possess considerable altitude, a pure and equable climate, a large amount of sunshine, and a dry atmosphere. Long sea-voyages often yield admirable results, and answer well for nervous, sleepless patients, who bear high altitude and exercise badly. An altitude of much more than five thousand or six thousand feet had better be avoided, at least for a continued period. Many patients must gradually accustom themselves to increased altitudes; indeed, it is best for all to ascend by slow stages. A weak heart is a positive contraindication to change to a high altitude. Nervous symptoms are also often aggravated. The emphysematous condition of the lungs developed in some persons by a high altitude prevents subsequent residence at sea level. Some improve in a cold climate, others in a warm one. A little observation will sometimes determine which to recommend. During the past season a patient was sent to a cold climate, because he had fewer "colds" in winter, and a general neurasthenic neuralgia, from which he suffered, was always better at that time of the year and from cold applications. His progress justified the choice. Previous sojourn in Southern California had not resulted favorably. Whatever change of climate is made, the patient must be impressed with this fact, *viz.*, *that a change of residence must be simply the means to a change in the habits of life.* The change in habits to consist of living in the open air for as much of the day as possible; under favorable circumstances, sleeping in a tent, and, if physically able, riding



horseback, eating plain, nutritious food, and securing all the sleep possible. This plan of treating phthisical patients, I have carried out for many years, and with the most gratifying results.

"When shall a consumptive be confined to bed?" is a question concerning which there is a great diversity of opinion and practice. Much depends upon the weather. If sufficiently warm and dry, a considerable portion of each day should be spent in the open air, even if it is necessary to carry the patient out and keep him there in a cot. Whether in the house or out of doors, the patient, with a maximum daily temperature above 100° F., should be at rest. The higher the temperature the stronger the indication for complete rest in bed. The advice to keep patients out of doors a great deal is given by many authors without the especial cautioning to avoid exercise. Rest is one of the most important factors in the treatment of phthisis. This has long been taught by the author, and has been recently emphasized by Dr. Mays. In our cold and changeable winter climate even advanced consumptives with cavities, high temperature, severe cough, etc., will often improve considerably if kept constantly in bed. Many an advancing case, in this manner, may be arrested and held over the season, when the bed and the patient may be safely taken into the open air. With the reduction of the temperature well below 100° F., a little exercise may be attempted, and further efforts governed by the result. How frequently do we find phthisical patients about the streets with the temperature as high as that attending pneumonia or typhoid fever, because the doctor has said: "Keep out of doors."

**CLOTHING.** The patient should during the entire year wear garments composed entirely or in part of wool, the weight being varied with the season. There are many reasons why the skin should receive special care; but on account of their feebleness, phthisical patients are often unable to bestow upon the skin the attention which it demands. Warm baths should be given sufficiently often to secure cleanliness, and light sponging with cold water should be daily employed for all except the very feeble. Reaction should be promptly secured. The bugbear of cold should not prevent the employment of this item of treatment, for with proper care it can be made use of without any bad result.

Confining occupations, or those attended by the inhalation of irritating particles or injurious odors, are prejudicial, as are all forms of work favoring a stooping position, or which in any manner interfere with the free action of the respiratory organs.

Especially in the early stage of phthisis, after the subsidence of acute symptoms, much may be gained by various methods calculated to stimulate the nutrition of the respiratory muscles and to expand the lungs. To secure this end the individual should carry out methodical breathing exercises three times daily, and the nutrition of the chest-wall

be improved by means of massage, friction, cold spongings and faradism. To favor the expansion of the apices the patient should practise the habit of carrying the shoulders well back, and cultivate the respiratory efforts.

**DIETETIC TREATMENT.** Careful attention to the food of phthisical patients is of the first importance. Food must be adapted to the digestive peculiarities of the individual. Idiosyncrasies must be considered. Most patients do well upon a broad and very liberal dietary, if there is appetite and digestive ability enabling them to take it. If not, every effort must be made to stimulate them. Change of food, of scene, and best of all, a change of air, are helpful. Generally it will be found acceptable and convenient to give alternately moderate meals of solid food, and liquid luncheons, *i. e.*, three meals of solid food, and three liquid luncheons daily. The most acceptable time for the luncheon is about an hour after food, when the sense of repletion has subsided, and before the appetite for the next meal can be affected. If the digestion is feeble, good results often follow an entirely liquid diet, or one containing a very small amount of solids. The best single article of liquid food is milk, if acceptable to the patient. The best form in which to administer it, is koumiss, which can be made at home by any intelligent person. When liked and well borne by the stomach, from four to six pints may readily be taken daily with advantage. This plan often corrects most distressing dyspepsia and improves the patient generally. Some prefer peptonized milk. While a large quantity of nutritious food is usually required for the best results, in many cases it is necessary to proceed with caution, giving small and frequent meals, and increasing gradually the quantity administered. When vomiting is frequent, and especially if there is catarrh of the stomach, daily gastric irrigation, followed by a quantity of predigested food through the tube, is an excellent practice. Many patients object very little to this procedure. In a case seen recently with Dr. Bartlett, a woman of fifty years of age, with fibroid phthisis and gastric catarrh, with some dilatation of the stomach, had vomited large quantities of ingesta nearly every evening for the preceding two months. These symptoms not yielding to a varied treatment, were relieved after the second irrigation. This improvement was followed by increase in flesh and strength. A large percentage of persons suffering from phthisis are benefited by the judicious and persistent administration of large quantities of food. It often requires tact to secure the taking of what is advised, and judgment to avoid disturbing the stomach. Animal food is particularly valuable and may be given in any form acceptable to the patient. Many thrive upon the use of really immense amounts of meat. Some of the most brilliant results which I have witnessed in the treatment of phthisis have resulted from a protracted residence in a tent among the foothills of the Rocky Mountains, the patient living largely upon venison. Fats

have a high reputation as food for the tuberculous. A careful review, however, of experience with this food has not impressed me with its clinical value, unless we may ascribe the benefit growing out of the free use of milk largely to the contained cream, which is doubtful. But it undoubtedly occupies a prominent place in the dietary of phthisis in the estimation of our best observers, and may be given in the form of cream, butter, fat meat, and the various oils, most prominent of which is cod-liver oil. The latter agent has had a long and unjustifiable popularity; still, it is an easily assimilable fat, and if not too much purified, possesses some medicinal influence.

Predigested foods are of occasional value in very feeble conditions of the digestive apparatus, but are probably too much used, especially the various patented preparations, which are especially objectionable in the treatment of young children, often developing a condition similar to scurvy. Grapes in large quantities have been given to phthisical patients with the belief that they exercise an important influence upon the disease. Lebert recommends half a pound of grapes at 7 A.M., at 5 P.M., and in a few days, a third half pound at 11 A.M. The amount taken at each time is gradually increased to one pound. The general diet prescribed at the same time is to be light and unstimulating. There is no doubt that the grape-diet acts well in some cases, and is a pleasant variation in food. It is recommended that patients rinse the mouth with a little soda and water after the meal of grapes. At certain European resorts, such as Montroux, the grape-cure is extensively practised during September and October.

Alcoholic stimulants are often valuable, but it is better to avoid them if possible. Malt liquors often favor an appetite, and increase of weight. Feeble digestive power and circulation, are usually benefited by the judicious use of the stronger alcoholic beverages. Occasionally cases are met in which prompt and continuous improvement has followed the daily use of quite large doses of whiskey. The risk attending the treatment is too great, however, to allow of its recommendation, except in carefully selected cases.

**SPECIFIC TREATMENT.** A variety of specific methods of treatment has been advocated, the most important of which is Koch's treatment by tuberculin. This agent is a 50 per cent. glycerin extract of cultures of the bacillus tuberculosis. The administration is hypodermatic, and is followed by both general and local reaction. The local changes are best studied in lupus treated by this method. Within a few hours of the injection, the diseased area develops redness and swelling, the color changing later to a brownish red. The mass becomes necrotic. The general symptoms consist of a chill, rise of temperature varying from 102° to 105° F., pain in the limbs, great weakness, and nausea and vomiting. The cough is usually increased. The results of this treatment



have been favorable in some cases of early pulmonary tuberculosis, while it has greatly aggravated others. The first serious check to the tuberculin treatment was received when Virchow published the results of post-mortem examinations upon twenty-one persons who had been subjected to the injections. The unfavorable results observed may be briefly enumerated as intense hyperæmia of the lungs, hæmorrhagic infiltrations and inflammatory processes in these organs, pleurisy, disseminated development of miliary tubercles, etc. Virchow also called attention to the possible perforation of intestinal ulcers due to the necrotic process. This publication was the beginning of a reaction against the method, which has been supposed by most persons to have overwhelmed it. Not so, however, the use of a smaller dose (one not sufficient to cause necrosis, and thus free the bacilli) and of purified tuberculin, has given better results, and it is possible that this agent may yet be demonstrated to possess great therapeutic value. Until sufficient experience has been obtained to enable the formulation of explicit instructions, its use should be limited to those thoroughly qualified.

**MEDICINAL TREATMENT.** It is probable that medicinal remedies do not possess a specific influence upon tuberculosis, *i. e.*, upon its specific, exciting cause, the bacillus tuberculosis, but rather upon the general nutritive condition of the patient, which they improve, thus giving to the system a greater power of resistance. For the treatment of many of the groups of symptoms it is necessary, in order to avoid repetition, to refer the reader to the various sections relating to the same occurring as independent forms of disease, more especially to those treating of bronchitis, pneumonia, and various disorders of the alimentary tract.

Certain medicines are popularly believed to be of special value in tuberculosis, regardless of the anatomical location of the lesion, the most prominent of which is *creasote*. The internal use of this remedy for tuberculosis has been so general, really occupying the first place of late as a specific remedy, that it is now possible to make a fair estimate of its therapeutic value. Several careful reviews of the subject have recently appeared, and these unite in denying any specific value to creasote. Its special beneficial action is upon the digestive tract. It possesses little or no influence upon the fever, and there is no proof of direct action upon the tubercle bacillus.

*Guaiacol* possesses an advantage over creasote in its lesser tendency to excite gastric disturbance.

The *hypophosphites of lime, soda and potash* have been in great repute for years as essential remedies against tuberculosis. Their popularity is due largely to the writings of Churchill, who employed them upon a most extensive scale. They have been generally used in combination, but the advocates of these preparations seem of late inclined to favor the employment of the hypophosphite of lime, or the hypophosphite of soda,

alone, or these two combined. The first decimal trituration of the hypophosphite of lime appears to the writer to be the most valuable preparation of this group, and if administered in the early stages of the disease, and for long periods, is of great value. It is most useful in young subjects with delicate tissues, and in scrofulous children. It is seldom followed by good results after the presence of pronounced septic symptoms. The cough is slight, the temperature subfebrile, the cheeks a little flushed, and there is a tendency to sweat. Much cough, fever or activity of symptoms generally contraindicates its use.

*Strychnine* is at present upon a wave of popularity as a remedy for pulmonary phthisis. Mays advocates its long-continued use in large doses. This observer considers it the most important medicine in this disease, and advises beginning with moderate doses—say, one-sixtieth of a grain—and increasing the dose to the point of toleration. I cannot recommend these large doses, but have seen, occasionally, excellent results from the use of one-grain tablets of the second decimal trituration, repeated three times daily. Strychnine appears to be of greater value in cases characterized by feeble digestive power and general neurotic symptoms.

*Iodine and its compounds.* *Iodine* has had quite a reputation as a remedy in the early stage of phthisis, being recommended by the older prescribers for cases presenting loss of weight in association with a good appetite. Nankivell and Hughes both sanction this statement. Personally, I am of the opinion that this is rather theoretical than based upon actual successful experience. In cases attended by dry harassing cough, iodine often acts extremely well, especially if applied locally over the affected lung. Used in this manner, it acts not only specifically, but as a counter-irritant. The *iodide of antimony* is undoubtedly the most valuable form in which to give iodine in phthisis. In three-grain doses of the second decimal trituration, repeated several times daily, it exercises a marked influence upon the cough and the profuse muco-purulent expectoration. Attending fever is generally moderated and the patient's condition as a whole much improved. *Iodide of tin* is nearly as valuable as the previous remedy, and like *stannum*, is pre-eminently indicated by profuse secretion rich in pus-cells, consequently yellowish or greenish, sweet, homogeneous, and usually easily expectorated.

**COUGH.** This symptom, which is one of the most troublesome features of the disease, must never be treated in a routine way with "cough medicine," but carefully studied in its every feature and prescribed for intelligently. Much may be accomplished by general management of the patient independently of medicine. The habits of the person, especially during the period of the twenty-four hours in which the cough is aggravated, should be inquired into. This will often reveal the exciting cause of the severe character of the cough. If it is aggravated during

the evening, see to it that the patient's surroundings and actions during the latter portion of the day are of the best. Often, especially during the evening, the living-room is poorly ventilated by reason of the closing of the doors and windows, the congregation of the family, perhaps of company, the smoking of tobacco, etc. Added to this there is increased talking upon the part of the patient, consequently increased excitement. These conditions not only exercise an unfavorable influence upon the cough, but must increase the patient's temperature and restlessness. Added to this the patient is often allowed to walk up stairs to bed, disrobe himself, put on a cotton nightgown, and lie down between cotton sheets, with the result of a severe coughing period at bedtime. All this should be changed. A quiet restful evening in a well-ventilated room, a little warm food, early retirement to the bedroom, if possible without ascending stairs, assistance in disrobing, a flannel nightgown, and a warmed bed, if the weather is cool, will materially diminish the cough. Many of the indications as to time of aggravation of cough, so highly prized by certain prescribers, are valueless in therapeutic results, because contingent upon gross violations of the kind enumerated. Night cough is quite apt to follow upon an aggravation during the evening or at bedtime. In many cases, however, it is the result of accumulated secretion, which the patient must be encouraged to expectorate at intervals. The sleeplessness and restlessness, largely dependent upon exhaustion, are also prominent causes of a night cough. A little liquid nourishment at frequent intervals during the night is helpful under these circumstances. Better results are often obtained by combining a small quantity of some alcoholic stimulant with the food. Under these circumstances the condition of the stomach should be investigated. A common error is to permit too heavy a meal of solid food in the evening when the patient is exhausted; such food remaining in the stomach undigested may excite night cough and is often vomited during the coughing paroxysms. If the patient is able to rest fairly well during the night the secretion accumulates in the bronchial tubes, or in cavities if they exist, and must be expectorated in the morning. The morning cough is therefore a prominent and necessary feature of most cases of pulmonary tuberculosis of the advanced form. Such a cough must be encouraged, cautioning the patient to endeavor to expectorate the accumulated matter with the least possible amount of coughing. If the patient is feeble, it is wise to endeavor to give some warm liquid nourishment during the latter portion of the night. A little whiskey in milk at this time is often wonderfully helpful, but it is a remedy which should not be abused. The day cough is affected very much by the habits of the patient, *i. e.*, increasing with exertion, and diminishing with quiet. The most prominent day aggravation is that after meals. This is due as much to the circumstances of the food-taking, as to the



presence of the food in the stomach. When this is a troublesome feature of a case, the patient should remain in recumbency for some time prior to and after eating, the food being taken in this position. It may be necessary to feed the patient. By this and similar methods readily suggested, the troublesome cough after meals, so often attended by vomiting of the food taken and consequent impairment of nutrition, may be prevented. In spite of the employment of such measures this form of cough may continue a most aggravated one. Under these circumstances the pharynx and accessory cavities should be examined, and any catarrhal condition or morbid sensibility should, if possible, be overcome. An irritable pharynx is a common cause of cough after eating. The diet should be carefully scanned in these cases. If there is an irritable pharynx or stomach, good results sometimes follow the taking of small pieces of ice during the fifteen minutes prior to a meal, the same plan being often successful immediately after meals. In all forms of cough associated with phthisis the nasal chambers, the naso-pharynx, the pharynx and larynx, should be carefully inspected, and catarrhs, ulcerations, hypertrophies, etc., treated by the simplest measures which will prove efficient.

**FEVER.** Patients with much elevation of temperature should be at complete rest in bed. This does not apply to subfebrile temperatures, *i. e.*, below 100° F. Complete rest in bed does not necessarily involve confinement to the house, which should never be permitted, unless the weather is cold or inclement. When circumstances make it possible, the patient should be carried out of doors and allowed to remain in the open air most of the day. The influence of change of climate, even to near points, upon the temperature is usually marked. In Philadelphia we secure excellent results for a time by sending patients to the seashore or into the mountains of Pennsylvania. Frequent changes are beneficial to some cases. There are few in which a judicious change of air will not result in a depression of the temperature. The skin should be kept in good condition, and during the acme of the fever cold spongings may be employed, or, in some cases, the cold bath. Rest, as an element in the reduction of temperature, has been suggested.

*Baptisia*, originally recommended by Dr. J. S. Mitchell for the fever of phthisis, is a remedy of considerable power. A tincture, prepared from the green plant, should be used, three to five drops every two to four hours. The lower dilutions often suffice. The *arseniate of quinine* has been more regularly efficient in my hands. It is best administered in tablets of the second decimal trituration every two to four hours. *Arsenicum* sometimes moderates the fever when its peculiar symptoms are present. *Silicea* has been helpful in cases associated with cavities, a large amount of purulent expectoration, and general sweats. *Ferrum phosphoricum* is suitable to the early fever of phthisis, but seldom of value after cavities and septic symptoms appear.

The old routine remedy, *quinine*, is now considered of but trifling importance. The more recent tar products, viz., *acetanilid*, *phenacetin*, etc., are now much employed, and possess a remarkable and positive influence upon the fever, but are not to be recommended for general use. The certainty with which they act is such that it favors a neglect of other remedies and of preventive measures. These preparations are given in doses of about three grains. If the fever does not yield to such doses it is rarely subdued by larger ones.

DISORDERS OF THE GASTRO-INTESTINAL TRACT. Few cases of phthisis recover in which the digestive function is feeble. The causes of such a feebleness vary much. Gastric catarrh is a common cause, but in a larger number it is the result of the general impairment of nutrition and consequent exhaustion. Nausea, vomiting, pain, flatulency, and diarrhœa, one or all may be present. Gastric feebleness often persists as the result of the continued use of solid food when such articles cannot be digested, or in exercise when it cannot be tolerated, especially near the meal time. In all cases of stomach irritability, the fever, if possible, should be controlled, and a carefully selected liquid diet administered. Koumiss in large amounts is most generally useful. A milk diet in some form may be continued for weeks with a gradual return to meat, eggs, bread, and later, the more general articles of diet may be added as they are proven acceptable to the patient's stomach. The diet should be rigidly prescribed in writing, with reference to the character, quantity, and the intervals of administration. *Pepsin* is of little use. A grain or two of *papoid* after eating often affords prompt relief and may be continued for some time with advantage. In acid conditions with fermentation and diarrhœa, *hydrochloric acid* may afford relief. *Arsenite of copper* often controls the nausea, vomiting, cramps, and diarrhœa, which are the product of indigestion. It is best given in the second decimal trituration, a short time before all food. When the presence of undigested matter in the stools is the most prominent feature of the diarrhœa, *cinchona*, *ferrum*, *ferrum phosphoricum*, or the *ferrum arsenicosum* may be considered. *Arsenic* is more suitable to the troublesome diarrhœas incident to intestinal ulceration. Drop doses of Fowler's solution, frequently repeated, sometimes help when the ordinary preparations of arsenic have failed. A few teaspoonfuls of burned brandy, frequently repeated, exercise an influence, which is beneficial if diarrhœa is present and persistent, and an astringent is indicated. Opiates are seldom of value and often harmful on account of the resulting retention of irritating matters in the intestines. In cases of diarrhœa not yielding to ordinary treatment, and particularly such as present the evidences of intestinal ulceration, irrigations of the rectum and colon may be employed every twenty-four hours. For this purpose boiled water answers well, but some prefer boracic acid, carbolic acid, or other germicidal substances.

**SWEATS.** The sweats of phthisis constitute a most important and sometimes even a dangerous feature. When demanding a special remedy for their treatment, it is best to administer *agaricine*, unless there are symptomatic indications for another medicine. The author was one of the first, if not the first, to teach the great value of this remedy in the treatment of this symptom. It is successful in the majority of cases in the first decimal trituration, one grain being given at bedtime. A single dose of this character is often followed by disappearance of the sweats for from one to three nights. Occasionally, it is necessary to give two or three doses during the evening or early part of the night, and sometimes the remedy must be repeated during the day. In a small percentage of cases the remedy fails entirely. The influence upon the feeble circulation, restlessness, sleeplessness, etc., is usually pronounced. If *agaricine* fails, it is best to administer *silicea* in the sixth trituration. With this medicine one may often control profuse, general night sweats after the failure of better-known medicines. *Phosphoric acid* may be useful when the general conditions indicating this medicine are present, and the same may be stated of *arsenicum*, *cinchona*, and *mercurius*. *Pilocarpine*, in the second decimal, has given excellent results in some cases of drenching sweats occurring in connection with rapid pneumonic phthisis, even after the failure of *agaricine*, *atropine*, etc. The *arsenate of iron* is effective in some cases having pronounced anæmia, and the symptoms of general deterioration more pronounced than those indicative of the lung-lesion. The first decimal trituration should be given three times daily. In obstinate cases, with a serious degree of prostration following the sweats, we may make use of *atropine* in doses of one one-hundredth to one-sixtieth of a grain, administered at bedtime, preferably hypodermatically. This remedy is usually effective for a time, although it often annoys the patient by reason of the peculiar symptoms liable to follow its use. *Picrotoxin* is recommended by some observers, and is to be given in the same manner as recommended for *atropine*. In extreme cases, in which the patient sometimes falls into a collapsic state, following the sweats, I have not hesitated to bathe the skin with a saturated solution of alum in a mixture of equal portions of water and whiskey. *Oxide of zinc* has considerable reputation for night sweats, in doses of two and one-half grains. *Camphoric acid*, also, in thirty grain doses at bedtime. Some recommend *strychnine*.

**HÆMOPTYSIS.** The early blood-spitting of phthisis seldom requires special treatment. The amount of blood lost is usually small, and if treatment has been carefully prescribed, it may be unnecessary to interrupt its course. If the patient's general condition is good, and there is excitement attending the hæmoptysis, a few doses of *aconite* will prove useful. If the blood-spitting does not quickly subside, *geranium*, which is highly commended by Dr. Snader, has in my own experience proven



a most efficient remedy. It is most effectual in doses of several drops of the tincture, which may be frequently repeated.

If blood-spitting is persistent, it is necessary to employ more active measures. In all cases the patient should be put at *perfect rest* in a cool room, prohibited from talking, should receive only cold food, and that in a liquid form. If the extremities are cool, hot bottles may be applied, and if good reasons are not apparent for the selection of some other medicine, *hydrastis* may be administered in ten-drop doses of the tincture or the fluid extract. This remedy has proven more effectual in my experience than either the much used *ergot* or *hamamelis*. If the bleeding area can be detected, it is recommended by many that an ice-bag be applied. It is better to avoid this if possible. Counter-irritation over the same surface appears to be almost as effectual and entirely unattended by risks of any kind. Dangerous hæmorrhage, uncontrolled by these means, or other well-indicated treatment, should receive hypodermatic injections of *morphine*. This is rarely required, however, except for the hæmorrhages occurring late in the disease. For adults, doses of a quarter of a grain, repeated two or three times in the twenty-four hours, are necessary. Unless full doses are given, this treatment is inefficient. *Atropine* has been recently much advocated, being given to the point of producing its physiological action. Persistent bleeding, associated with feebleness of the circulation, is often favorably influenced by stimulants.

**PAIN IN THE CHEST.** Pain in the chest is most frequently myalgic, and the product of malassimilation and the strain attendant upon coughing, conditions tending to the development of irritable muscles. Patients, and too many physicians, are inclined to locate the lung lesion by means of the seat of the pain. While parietal pain occurs more frequently over the affected than the sound lung, the localization of the lesion by means of pain is an error, unless such pain is pleuritic, and even in the case of pleurisy it is not an infallible method. The greater number of attacks of acute pain occurring in phthisis are due to pleurisy; the smaller number to intercostal neuralgia. Quiet, and the use of *aconite*, *bryonia*, *kali carb.*, *scillitin*, or *sulphur*, will prove promptly remedial for the pleurisy. The intercostal neuralgia is more troublesome to combat, but is sometimes relieved by *aconite*, or better, the *nitrate of aconitine* (grain doses of the third decimal trituration repeated every three hours), less frequently by *gelsemium*. Counter-irritation with iodine, also the galvanic current about the painful foci, sometimes helps. For the myalgia it is necessary to improve the nutrition, lessen the cough and improve the state of the chest-muscles by means of bathing, frictions, massage and breathing exercises. The most important remedies are *bryonia* and *actea*.

In severe attacks of myalgia prompt relief can generally be secured by the use of grain doses of *acetanilid*, repeated every half hour, until the pain is controlled.

INSOMNIA. The sleeplessness occurring in the course of phthisis is generally the direct result of other conditions which are troublesome at night, such as cough, fever, etc. It follows that the rational treatment lies in the relief of these symptoms. Before, therefore, prescribing specifically for sleeplessness, it is necessary to examine into the cough, fever, the condition of the digestive organs, etc. Spongings of the entire body with water at the most agreeable temperature, of the spine with water as hot as it can be borne, cold applications to the head, a hot foot-bath, a weak mustard plaster to the epigastrium, are simple remedies of occasional value.

LOCAL TREATMENT OF THE RESPIRATORY TRACT. Local medication is as justifiable in the treatment, especially of advanced phthisis, as it is in the treatment of wounds by the surgeon. When we consider the extensive tubular and cellular structure of the respiratory apparatus, and the inflammatory changes attacking them, with their destructive termination, it is evident that antiseptic measures must be beneficial, and this is confirmed by experience. The method employed by the author is substantially that advised by Dr. Austin Flint, Jr. It consists in the inhalation, without effort, for one hour in each twenty-four, of various drugs possessing antiseptic properties. Unquestionably the most important of these is pure *beechwood creasote*, which should be mixed with equal quantities of alcohol and chloroform. The chloroform may sometimes be omitted with advantage, but should be replaced with an equal quantity of alcohol. For one hour's inhalation one drachm is required; but, to avoid waste, twenty drops should be added each twenty minutes. *Guaiacol*, *terebene*, *eucalyptus*, and other well-known substances may be used in the same manner. An inhaler constructed of fine wire-cloth, and containing a sponge or absorbent cotton for the reception of the inhalant, may be used for this purpose. If not procurable from a dealer, such an inhaler can be readily manufactured out of light wire, covered with cheese-cloth, and is specially satisfactory during warm weather. The inhalations have a sedative influence upon the cough.

TREATMENT OF CAVITIES. Following the advice of Pepper, the writer has for some years treated cavities of considerable size by means of the injection of antiseptic solutions, especially of iodine, creasote and creoline. The injections have been made from one to three times weekly. In some instances there has been a high degree of improvement, especially in very chronic forms of phthisis with large cavities. The most prominent result has been a lessening of the expectoration and the cough, and, associated with this change, a reduction in the temperature and a general improvement in the health. Only one unpleasant result has followed this practice, viz., the development of pneumonia, following rapidly upon a creolin injection. Whether the injection excited the pulmonary inflammation or not, is a matter of conjecture. The patient

recovered, apparently none the worse for the pneumonia. The injections are administered with the ordinary hypodermic syringe, to which must be fitted an extra long needle. A weak solution of the selected drug must first be employed, and the strength gradually increased. Accurate diagnosis is, of course, essential to good results.

### LARYNGEAL TUBERCULOSIS.

**Etiology.**—Laryngeal phthisis embraces those lesions incident to the localization and propagation of the bacillus in the larynx. It may develop as a primary affection of this organ, or it may appear secondarily to pulmonary tuberculosis. Most cases develop after pulmonary phthisis is well advanced. The primary variety is rare; some even doubt if a laryngeal phthisis ever precedes the development of the pathological process in the lungs. Inability to discover the physical signs of involvement of the lungs cannot be taken as evidence of the primary character of the laryngeal affection. Only exhaustive autopsies can be accepted as conclusive evidence. Many cases of supposed primary laryngeal tuberculosis have been demonstrated by expert diagnosticians to have succeeded limited pulmonary consolidation. Rapid extension of the laryngeal lesion before discovery of the disease of the lungs, which is claimed to be evidence of primary disease, proves nothing, as it is not an uncommon observation in well-developed phthisis that the progress of the later-developed trouble in the larynx is much more rapid than that of the primary pulmonary lesion. At this time, there is under my observation a case of phthisis of six years' standing. Now the slight pulmonary lesion is latent, but within five months there has developed a laryngeal involvement which has progressed until it is now characterized by extensive ulcerations. The patient's only complaint is concerning the condition of the larynx. The common conception of its origin is that a catarrhal larynx becomes infected by means of the sputum (auto-infection). It seems consistent with our knowledge of tubercular infection in other portions of the body, to believe in direct infection of the larynx from inspired bacilli. It is very rare that the laryngeal lesion is seriously advanced before there are evidences of lung disease. Of 1,226 post-mortem examinations of tubercular subjects, reported by Heinze, from the Pathological Institute of Leipzig, 30.6 per cent. manifested laryngeal lesions. Males are attacked oftener than females, probably for the reason that men are more exposed to the causes of laryngeal catarrh, which offers a good soil for the development of local tuberculosis. Most cases occur between the ages of twenty and thirty-five years. J. Solis-Cohen has reported a case in a child of seven months.

**Pathology and Morbid Anatomy.**—The essential pathological element is the tubercle nodule, which undergoes caseous degeneration



and ulceration. The primary change is in the nature of a laryngeal catarrh. The mucous membrane is observed to be swollen, pale, and soon are here and there to be seen minute tubercles, which are in close relationship to the bloodvessels. Opinions differ as to the frequency with which these tubercular granulations are visible. Closely united groups of tubercles finally undergo caseation and destruction. The resulting ulcers are rounded or irregular in shape, and vary much in size. The inter-arytenoid region is the one most frequently attacked. Sometimes but one side of the larynx is involved, and in cases in which but one lung is diseased, the side of the larynx affected may correspond to the diseased lung. The depth of the ulcerations, like their area, is subject to great variations. If deep, a peculiar funnel shape is quite common. The ulcerated surface is grayish or yellowish. The thickening of the mucous membrane is most conspicuous upon the arytenoids, and results, when fully developed, in a peculiar pyriform enlargement of the aryepiglottic folds, one or both of which may be attacked. The epiglottis is frequently involved, and may be largely destroyed. It may be so much enlarged as to obstruct the view into the larynx. It may be distorted, and assume various shapes. The ulcerations, which extend in area rather than in depth, are most frequent (according to Heinze) upon the vocal cords, the arytenoid bodies, and the epiglottis, both cords being affected in the majority of cases, which may be cut from the vocal processes of the arytenoids, or their muscles from their insertion. The false bands are seldom attacked. Less frequently ulcers are present over the arytenoid cartilages. "Those which lie toward the base of the cartilages are almost always bilateral. They are seldom, if ever, visible in the laryngeal mirror, and have a peculiar tendency to penetrate deeply into the tissues so that they often reach the perichondrium, and lead to necrosis of cartilages" (Fagge). The epiglottis is ulcerated about as frequently as the arytenoid body. The laryngeal surface is most frequently involved. If the upper surface of the epiglottis is attacked, the ulceration may extend to neighboring parts. In many, ulceration will progress until extensive destruction of soft tissues and cartilages has taken place. Papillary excrescences are not uncommon upon the edges of ulcers. The mucous and submucous tissues are thickened. The surface of the mucous membranes may be pale, and present the prominences due to the tubercle nodules. The changes frequently overstep the laryngeal bounds, and involve the epiglottis and pharynx above, and less frequently the trachea and bronchi below. Perichondritis often leads to serious results. Intercurrent œdema may develop stenosis at any time. The virus usually enters by the lymph vessels, and the early infiltration involves the parts subjacent to the epithelium, the epithelium appearing normal until ulceration develops, the destructive change may be purulent, or a simple breaking down of the unorganizable products.

**Symptoms.**—The onset is insidious. The most important symptoms are progressive impairment of voice, cough, dysphagia, dyspnœa and pain. The impairment of the voice varies from the slightest huskiness in the early stage to complete aphonia, with the development of extensive laryngeal changes. The quality of the voice often suggests the nature of the affection. Ulceration is not the only factor concerned in the production of the hoarseness, inflammatory swelling of the tissues and weakened muscles exerting a decided influence. The more the vocal cords or the tissues in their immediate vicinity are thickened or ulcerated, the greater will be the hoarseness or aphonia. A slight thickening or ulceration affecting the cords in the early stage will lead to decided hoarseness, while lesions involving other regions of the larynx may continue to a fatal issue without material alteration in the voice.

Cough is usually troublesome, and in the beginning may be largely symptomatic of the underlying pulmonary disease. With the development of the laryngeal complication, however, it undergoes more or less change in character. With extensive changes the cough becomes husky. It may be spasmodic. The expectoration is more or less purulent, and may be blood-streaked, and contain elastic fibres. Difficulty in swallowing constitutes one of the most distressing symptoms of the disease. It appears when the ulceration has extended to the epiglottis or pharynx. This symptom interferes greatly with the taking of food. In the latter periods of the disease the cough usually becomes frequent and very annoying, causing much pain, and often vomiting. The involvement of cartilages, with consequent necrosis, an occasional œdema, or invasion of the crico-arytenoid articulation, leading to symptoms suggestive of paralysis of laryngeal muscles (Holmes), add to the symptomatology.

Pain in the early stage varies; slight painful sensations are complained of, and there may be some distress during swallowing, due to pressure upon the diseased organ. The pain may radiate to the ears. With the increase of destructive changes the pain increases, and reaches its highest development when exposed ulcers appear upon the epiglottis. So great are the pain, choking, and the resulting regurgitation of food under the circumstances, that interference with alimentation is not uncommon.

The general symptoms are those of pulmonary phthisis. Death may occur from exhaustion, or rapid termination may take place, due to œdema of the larynx. The progress of the disease is variable. An acute tuberculosis of the larynx may terminate within a few weeks, or at most in a few months; while chronic ones may continue for several years, often with very little change for long periods of time.

A very acute form of tubercular laryngitis has been described, in which an acute miliary tuberculosis attacks the larynx and neighboring parts, viz., the pharynx, tonsils, mouth, etc., rapidly progressing to ulcera-

tion, and a termination in six or eight weeks. As a miliary tuberculosis the process may begin above, and extend to the larynx secondarily. Cases of this form have been reported cured by the use of the curette and lactic acid.

**Laryngoscopic Appearances and Diagnosis.**—Inspection with the laryngoscope is essential to a correct recognition of the condition present. In the early stage of the disease the appearances are those of an ordinary catarrh. Great stress was laid by Semeleder upon a paleness (anæmia) of the larynx as a forerunner of laryngeal tuberculosis, but this anæmia can hardly be regarded as more than a feature of the constitution which so often precedes the outbreak of laryngeal tuberculosis. The important diagnostic feature, however, of established laryngeal phthisis is the presence of a characteristic ulceration. The peculiarities to remember are breadth, shallowness, grayish color, with a layer of mucus (not pus), margins which are not very distinctly defined, and not surrounded by an inflammatory areola. According to Fränkel the surface of these ulcers resembles sliced bacon. The presence of pulmonary phthisis, and especially of the bacilli of Koch in the secretions taken from the surface of the ulcers, makes the diagnosis certain.

A pyriform swelling of one or both arytenoid prominences is present in most cases. These are usually pale; but they occasionally present a red or livid appearance, and later, the ravages of ulceration. It is very easy to overlook small ulcers in the lower portion of the larynx. Such may be invisible or seen only in part covered with mucus. Late in the disease the appearance of the larynx is greatly altered. The partially destroyed vocal cords, extensive ulcerations and prominent swellings giving a peculiar image.

The separation of laryngeal tuberculosis from syphilis is often impossible. The greater tendency of syphilitic ulceration to involve also the pharynx, and to heal at some points while invading others, is suggestive. Also in syphilis a careful search will often result in the discovery of other evidences of the presence of the disease.

In the cases of reported cure there is often a great question as to the syphilitic nature of the lesion. The test of medication is sometimes necessary.

**The Prognosis** is always grave, but few recoveries having been reported. Still, results are improving, and the more energetic methods of treatment recently adopted have given a degree of success which leads us reasonably to hope for still better results. If the pulmonary disease is in control, and the lesions in the larynx few and small, some hope of a favorable result may be held out to the patient. A fatal result occurs most frequently when the epiglottis is involved, on account of the serious difficulty such a condition occasions in swallowing.

**Treatment.**—This affection merits most careful attention to its



treatment, especially in the early stage, which it seldom receives. Much negligence in the direction of prevention would be avoided, were practitioners to energetically treat all laryngeal symptoms arising in the course of pulmonary phthisis. When the taking of food is attended by serious difficulty, a four per cent. spray of cocaine, used a short time before eating, usually gives relief. The general care of the patient should be such as suggested for pulmonary phthisis. The larynx must be kept sufficiently at rest to prevent all irritation of the inflamed tissues from using the voice, a feature of treatment generally grossly neglected.

At first the local treatment should consist of a simple cleansing spray of some alkaline solution, such as *chloride of sodium* or *boracic acid*, ten grains to the ounce of distilled water. Various combinations have been used for the same purpose, but they do not appear to be preferable. In some rather indolent cases, sprays of the *peroxide of hydrogen* give good results. The cleansing may be followed by a spray of *menthol* in *vaseline*, thinning the solution by heat before using. *Listerine* diluted with two or three parts of water has also given good results. *Creasote inhalations* as recommended for pulmonary tuberculosis are often of service. If these means fail to make a favorable change, the use of *iodine* in the form of an alcoholic spray, or applied directly to the parts by an applicator, has given the best results in the author's hands. A gradual increase in strength up to a saturated solution, repeating the treatment every forty-eight hours, is the plan suggested. With proper apparatus and care these solutions can be used without causing spasm or other annoying results. Applications of dilute *lactic acid* (20 per cent. watery solution at first, and gradually increased in strength) first recommended by Kramer, have been highly recommended.

Heryng introduced the recent method of scraping away or excising the diseased tissues, a method which promised well, but can be practised only by experts. Reports showing the cure or relief of at least 50 per cent. of the cases under this treatment, have been published. Lactic acid is applied to the surface after the operation.

Dr. A. C. Peterson recommends watery solutions of *calendula*, "one to twenty or weaker, with the addition of two or three drops of *carbolic acid* to the ounce." The above is quoted by Ivins, and receives his commendation. Dusting the larynx with *iodoform* has been advised by Bosworth. This is a most valuable remedy. *Morphia*, locally, in watery solution, is often a great comfort to the patient. It must not be forgotten that its systemic action is readily gained in this manner.

*Pyoktanin* and other aniline dyes promise to be valuable. They may be used as a spray of the saturated solution, or applied directly to the surface with forceps and cotton.

*Food* should be carefully selected. Fluid foods are generally taken more easily if somewhat thickened. When swallowing is difficult, great

relief is secured by adopting the plan first suggested by Norris Wolfenden. The patient is directed to lie face downward on a couch, with the head hanging over, and take fluid nourishment through a tube, or even from a cup, while in this position. Swallowing is thus rendered painless.

The *oesophageal tube* is sometimes used with advantage when swallowing causes serious symptoms. Rectal alimentation is a valuable adjuvant. Tracheotomy has been advised to secure rest to the larynx; but most authorities reserve it for the relief of stenotic symptoms.

A large number of medicines have been recommended for laryngeal tuberculosis, and there can be no doubt of the favorable action of many, especially in the modification of symptoms for a time. In the initial period, *ferrum phosphoricum* sometimes suits the totality, and has proven efficient clinically. *Drosera* is advocated by Rene Sarrans as a prophylactic. As to the value of this medicine in early pulmonary phthisis I can testify. *Stannum iodide* has controlled the profuse sweetish expectoration, and favorably modified the laryngeal ulceration in some cases. The *iodide of arsenic* is called for when anæmia is pronounced and there are other marked evidences of constitutional deterioration. *Iodine* relieves a variety of symptoms, but especially those of a croupy character, which sometimes develop suddenly. *Apis mellifica*, *bromine*, *kali iod.* and *sanguinaria* are indicated for œdematous developments. *Mercurius nitr.*, according to Dr. Malcom Leal, has removed small laryngeal ulcers. Meyhoffer, whose experience with this disease has been large, places considerable dependence upon medicines, reporting the best results from *nitric acid*, *nitrate of silver*, *arsenic*, *iodine* and *seleniate of soda*.

Jousset recommends *drosera* for the severe cough, and *calcareo carbonica* for the ulceration. Bæhr recommends *manganum*; and Kafka *atropine*, in drop doses of the second decimal dilution, for the troublesome cough.

J. Solis-Cohen advises the use of *creasote*, stating it should be avoided while the temperature is high or pulmonary hæmorrhage is frequent. The opinion seems general that *tuberculin* should be avoided, although it is possible that further use of *tuberculocidin* will give better results.

The medicines recommended for the various forms of laryngitis should be consulted. The importance of change of climate must not be overlooked, great temporary improvement often resulting.

## TUBERCULOSIS OF SEROUS MEMBRANES.

There are instances of primary tuberculosis of the serous membranes. Usually, however, tuberculo-serous inflammations are but a part of a general morbid process.

SEROUS MEMBRANES WITHIN THE CHEST. Primary tuberculosis of the pleura is probably not as common as is generally believed, for the reason that tubercular lesions of the lungs and bronchial glands exist

frequently without detection, and even post-mortem examinations require great care, lest they be overlooked. One should report a pleurisy as primary and tuberculous only after a most painstaking examination after death.

The pleurisy attendant upon pulmonary phthisis may appear to be simple, *i. e.*, the inflamed tissues may not present the evidences of tubercles. Adhesions are constant. Destructive changes may occur at a small focus, and perforation result in pyo-pneumothorax. The tubercles may be miliary, large and caseous, gray or yellow. Attending effusions may be serous, purulent, or rarely bloody. The doctrine that all pleurisies are tubercular in origin cannot be supported, but the etiological prominence of tuberculosis in the causation of pleurisy is increasing.

TUBERCULOSIS OF THE PERICARDIUM is a rare affection. Most authors disagree with Hensch, who declares that tuberculosis is a frequent cause of pericarditis in childhood, and secondary to tuberculous disease within the chest. Miliary tubercles may be numerous, or caseous masses may be associated with a thickened and adherent membrane. The granulations may be easily overlooked. The focus to which this affection is secondary, may be in the lungs, the pleura, or the mediastinal glands. Pericarditis both acute and chronic may result.

The symptoms and signs are much the same as in other forms of pericarditis. It is differentiated usually by the evidences of extension from adjacent tuberculous organs. Occasionally it is impossible to make out the true nature of the case. In chronic tubercular pericarditis the clinical manifestations may be those of hypertrophy and dilatation of the heart.

TUBERCULOSIS OF THE PERITONEUM is usually a part of a general tuberculosis. It may be localized in the peritoneum. Miliary granules are often found on the peritoneum over tuberculous intestinal ulcers under these circumstances. The granulations may be traced along the line of lymphatic vessels. When primary it is an acute disease. The development is usually miliary. Cheesy, and particularly calcified, masses are occasionally present. The miliary tubercles are easily overlooked. It is best, therefore, to examine the omentum carefully if in doubt. Inflammatory changes often co-exist, leading to adhesions between the abdominal organs. All may be matted together in one mass. The exudate may be mainly fibrinous or serous, hæmorrhagic or purulent.

The clinical course is subject to great variations. A moderate abdominal dropsy, and peritonitis acute and chronic, are the most important manifestations. The effusion is not large, and there is danger of confounding this condition with hepatic cirrhosis or portal thrombosis, especially if the tubercle bacilli cannot be demonstrated. It is interesting in this connection to note that tubercular peritonitis is often a late complication of cirrhosis of the liver. Fever is a prominent



feature in the acute form, which is attended by abdominal pain, tenderness and distension. In protracted cases the symptoms may quite closely simulate a continued fever, especially typhoid fever. Unusual forms are those in which the onset and severity have been such as to lead to a diagnosis of hernia or enteritis. There are also very rare cases in which the symptoms are so slight as to fail to even offer a suggestion as to the possible nature of the case. There are still others in which pigmentation of the skin suggests Addison's disease as the cause of the trouble. Several pathological conditions peculiar to tubercular peritonitis simulate tumor: (1) A sacculaton of the exudation, it being confined within bounds formed by the adherent abdominal structures. When in the lower abdomen, such a development has been mistaken for an ovarian tumor. The same development may occur in the pelvis in association with tuberculous disease of the Fallopian tubes. (2) Gardiner, especially, has called attention to a puckering and rolling of the omentum, which results in the formation of a transverse tumor-like body in the upper abdomen. It is less common in peritonitis due to cancer. Changes in the omentum may also cause lateral tumors, especially in the right iliac region. Careful manipulation of the tumor in the umbilical region reveals length in excess of breadth. This is so marked that it has been spoken of as a "cord-like development." (3) Tumors formed by the thickened and retracted coils of intestine. A single loop or the entire gut, both large and small, may be involved.

It is convenient, in connection with this subject of tuberculous conditions simulating abdominal tumor, to mention tuberculosis of the mesenteric glands, which also gives rise to abdominal enlargements associated with ascites, distension of the abdomen with gas, and colicky pains, thus presenting points of similarity to peritoneal tuberculosis.

The most important feature in the diagnosis of these lesions is to be able to discover the bacilli, or trace a relation between them and previously existing tubercular disease. This requires a most careful investigation of the history of the patient, as well as an examination of all portions of the body.

### RENAL TUBERCULOSIS.

In tuberculosis of the urinary organs the lesions are rarely confined to the kidneys. It may be a primary or secondary development. As already stated, it is exceedingly difficult to positively assert that a case of tuberculosis is primary on account of the wide diffusion and obscurity of the lesions. A certain number of cases, however, are apparently primary. The causes of a primary localization in the urinary apparatus are not clear. The ordinary predisposing causes of phthisis have been arraigned, but they can only produce a catarrh which is favorable to the development of bacilli. The secondary tubercular form generally follows

upon primary disease of the epididymis, seminal vesicles, prostate gland, or from a more distant focus, the lungs. The disease has been met at all ages, but is most frequent between adolescence and the fortieth year. Men are oftener attacked than women. The infectious elements are probably conveyed to the parts by the blood- and lymphatic-vessels. The process usually begins in the kidneys and extends downward, the reverse of this being doubted by many authorities. The organs may be affected at numerous foci, or an almost continuous lesion may exist. Meckel states that the disease is often unilateral, the right kidney being the most frequently attacked. The condition of the non-tuberculous organ varies very much, it may be normal, carcinomatous, or necrotic. Cellular growth in the pyramids, followed by cheesy degeneration and softening, and the discharge of the detritus into the urine, represent the lesion as found in the kidney. The situation of the pyramids is occupied by a cavity with a cheesy wall. These cavities may contain large cheesy and calcareous masses. After destruction of the pyramid, the diseased process pushes on into the cortex, which becomes irregularly involved. In highly developed cases of tuberculous destruction of the organ, little remains but a thick-walled capsule, containing sacs and sinuses with walls consisting mainly of connective tissue. The weight of the organ is usually greatly increased. The pelvis of the kidney presents tubercular infiltration and ulcerated areas, detached fragments from which may occlude the ureter, resulting in distension of the kidney passages. The ureter is frequently changed into a hardened cord, which is irregularly dilated.

Involvement of the bladder is usually first observed at the fundus. Small ulcers, generally involving the mucous coat only, coalesce, forming large, irregular ulcers with elevated sharply outlined edges. It is not uncommon for a tuberculous ulcer in the bladder to become encrusted with inorganic matter, especially phosphates. The urethra is rarely attacked. The symptoms are essentially those of catarrh of the urinary mucous tract; sometimes they resemble a vesical catarrh, in other cases, a catarrh of the pelvis of the kidney. The urine is purulent and voided frequently (a suggestion of implication of the bladder), and is of an ammoniacal or offensive odor. The amount of urine may be increased. Its specific gravity remains about normal. Hæmaturia is occasional. The sediment consists of mucus, pus, very small masses of cheesy matter, wrinkled round cells, free nuclei and granular matter, elastic fibres and connective tissue fragments, epithelium from the various regions, triple phosphates, and, in the majority of cases, tubercle bacilli. In most cases the indications of a pyelitis predominate, and unless the bladder becomes involved, the patient may remain in a fair condition of health for many years. In a clearly demonstrated case in a woman of sixty-one years, under my care at present, the evidences of the disease

have existed for several years. During this period, the general health has permitted almost constant activity.

Involvement of the kidneys should be suspected, if in connection with some of the urinary conditions detailed, there is in a tuberculous subject, persistent pain in the region of one kidney. Pain is, however, often absent or developed by pressure only. More important is the presence of a fluctuating enlargement of the kidney. If the obstruction causing the retention and consequent hydronephrosis gives way, the clear urine becomes clouded. With advanced disease in one kidney, involvement of the second, or an increase of tuberculosis at other points, with fever, chill, sweats, etc., may occur.

The duration of renal tuberculosis averages about a year; but as stated previously, cases may be prolonged indefinitely. Recovery is possible. This has been shown by post-mortem examinations.

**Treatment** is of the most general character, and accomplishes but little. Tuberculin may possibly prove of use in selected cases. If the bladder becomes involved, surgical means may be necessary. Men suffering from tuberculosis of the prostate or epididymis should avoid coition for fear of infecting others.

### TUBERCULOSIS OF THE NERVOUS SYSTEM.

Acute tuberculous infection of the brain leads to the eruption of miliary tubercles in the meninges, especially at the base, causing a meningitis with effusion. This affection has already been described (see page 346). A less frequent form is found in a meningo-encephalitis, which is occasionally localized and excited by the development of small tubercles in the pia mater and in the walls of the minute arteries of the cerebral substance. It most frequently occurs during the course of pulmonary phthisis. The consequent disturbance of the circulation often leads to cerebral softening. The symptoms of this form depend much upon the location of the tubercular patch and the rapidity of development. Hemiplegia has been observed as a symptom.

A form known as solitary tubercle is met occasionally, especially in young subjects. This is an isolated tubercular development, varying in size from a pea to an orange, and found most frequently in the cerebellum. Occasionally the growth may be multiple, as many as a dozen or two being scattered through the brain. The lesion is roundish or lobulated, yellowish, dry and caseous. The border is grayish or pinkish. The adjacent brain-tissue may undergo softening. The centre of the tubercle may likewise undergo softening or calcification.

### TUBERCULOSIS OF THE BLOODVESSELS.

This occurs in tuberculous organs, especially in the lungs. Softened tubercles in the vessel-walls may lead to infection of the blood or rupture



externally with resulting hæmorrhage. Minute granulations develop freely in the walls of minute bloodvessels, especially those of the brain.

### TUBERCULOSIS OF THE LIVER.

Tuberculosis of the liver is usually part of a general tuberculosis. The lesion is composed of the minute gray granulations, or larger masses, bright yellow in color, many of which have softened, forming numerous abscesses, varying in size from that of a pea to that of an English walnut. These tubercles develop within the smaller bile-ducts, the staining of the mass with bile giving rise to the color referred to. Tuberculous masses up to the size of the fist are occasionally encountered secondarily to tuberculous peritonitis or perihepatitis. When tuberculosis of the liver is chronic, it is associated with hyperplasia of the connective tissue, and is called by some pathologists "tuberculous hepatitis." It is seldom that local symptoms accompany hepatic tuberculosis. The liver may be enlarged, its function disturbed and ascites may be present.

### TUBERCULOSIS OF THE ALIMENTARY TRACT.

Tuberculous lesions may occur at almost any point between the lips and the anus. Upon the *lips*, *tongue* or *hard* or *soft palate*, and the tonsils, the disease may appear first as a nodular prominence; but a developing ulcer often first attracts attention. The nature of the affection in this location is often in doubt, more especially, perhaps, on account of the rarity of the disease here. It is often mistaken for cancer and syphilis. Examinations of a fragment of tissue for tubercle bacilli, or inoculations from the sore, may be employed for purposes of diagnosis.

The *pharynx* is usually involved by extension from the larynx. Numerous miliary granules may be seen studding the posterior wall. There is often considerable ulceration, leading to pain and choking upon swallowing; and the suffering from this cause is considerable. Involvement of the *œsophagus* is exceedingly rare. The same may be said of the *stomach*, although it is more frequently attacked than is the *œsophagus*. Tuberculous ulcers of considerable size have been reported.

*Intestinal tuberculosis* is generally secondary to the pulmonary form. The source of the infection is the swallowed sputum. Primary involvement of the intestine occurs far more frequently in children, and is commonly due to the taking of infected food, especially the milk from infected cows. Rarely do cases arise from the extension of disease from the peritoneum. The tubercular growth begins in the lymph glands of the intestines, both the solitary follicles and Peyer's patches being involved. Fusion of the miliary granulations beneath the epithelial covering leads to a diffuse development. By extension the deeper structures of the intestine are gradually implicated, even to the peritoneal covering. Upon the internal, or mucous coat surface, destructive changes

gradually extend, until ulcers of considerable size are formed. These may be irregular; but extension is especially in the direction of the diameter of the gut, giving rise to the characteristic "girdle-like" ulcer. Ulceration is sometimes very extensive, involving large tracts of intestinal surface. Miliary granulations may be frequently seen with the unaided eye in the edges or the base of the ulcers. The region of the ileo-cæcal valve is most frequently attacked. Intestinal tuberculosis is often associated with similar changes in the mesenteric glands and peritoneum. Perforation with consequent peritonitis is a not uncommon sequence. The healing process may lead to stricture of the bowel.

The symptoms are not, as a rule, very pronounced, until diarrhœa makes its appearance; and in reference to this it is known that ulcers of large size are sometimes found upon autopsy, whose existence has not been suspected.

What appears to be a primary tuberculosis of the intestine is rarely developed in adults, and presents quite acute symptoms, such as fever, diarrhœa, pain, and, occasionally, hæmorrhage. This condition, subject to interruption, continues until the lungs manifest symptoms of disease, when the nature of the case at once becomes manifest. Before pulmonary complications are present, the degree of attending wasting and constitutional failure may have already suggested the nature of the malady.

In rare instances localization of the symptoms in the right lower portion of the abdomen may have suggested appendicitis. Disease of the ileum due to lesion of the ileo-cæcal valve produces the same group of symptoms. Primary tubercular disease of the abdominal organs in children was at one time designated *tabes mesenterica*, and this term is still found in many of our text-books. It consists in swelling and caseation of the abdominal lymph glands, which may also undergo purulent softening or calcification. The enlarged glands may often be felt through the abdominal walls. The liver also is frequently perceptible below the ribs. A variety of symptoms indicative of abdominal disturbance, viz., diarrhœa, distended tympanitic abdomen, etc., joined to general and progressive emaciation, anæmia, hectic fever, and the age of the patient, suggest the nature of the disease. Post-mortem examinations of such cases show tubercular involvement of the structures within the abdomen. The lungs may be normal.

A tuberculous abscess in the ischio-rectal tissues, which often results in anal fistula, is not uncommon in sufferers from pulmonary phthisis. So suggestive are these abscesses of the presence of tuberculosis, that their presence should inspire careful investigation of the patient's condition. Search for the bacilli in a piece of the tissue of the abscess wall, or inoculation experiments, will make the diagnosis clear.

## SYPHILIS.

**Synonyms.**—Lues venerea; the pox.

**Definition.**—Syphilis is a chronic, infectious disease, the virus of which can be inoculated on the healthy only by intimate contact over an abrasion of the skin, or a delicate mucous membrane; characterized, clinically, by an initial lesion occurring at the point of infection, and this lesion is followed in time by manifestations of the disease in various portions of the body. The lymphatic glands, the skin, the bones, the various viscera, and the entire nervous system, may suffer severely from the ravages of this disease. Syphilis may be communicated by heredity, in which case it is spoken of as *congenital* or *hereditary syphilis*.

**History.**—The origin of syphilis must be regarded as by no means established. By many, the disease is believed to have been introduced into Europe by the followers of Columbus on their return from America, at the close of the fifteenth century. By others, it is claimed that evidences of the disease in prehistoric ages may be found in widespread quarters of the globe. The existence of the disease in ages long since past, has been inferred mainly from the discovery of bones exhibiting lesions, which appear to be identical with or similar to those found in bones from cases in which syphilis is known to have existed. Parrot is an authority who believes that such evidence has thoroughly established the prehistoric existence of syphilis. Objections to this view have been offered. So far as is known, no bones, exhibiting the lesions of the hereditary disease in children, have been discovered, notwithstanding the fact that numerous specimens, exhibiting the changes due to non-specific osteitis and periostitis, have been found.

Much importance has been attached to certain ancient writings—those antedating the Christian era by centuries—apparently showing that syphilis was prevalent in those days. Such evidence cannot, however, be regarded as satisfactorily reliable, for most of these writings were in Asiatic tongues, now obsolete so far as modern usage is concerned. The translators were, necessarily, men skilled in other fields than medicine, thus greatly impairing the accuracy of their works. It must also be remembered that the peculiar style of language in those days was such as to cloud thought, especially when viewed by modern eyes.

Ancient medical writings, those of Hippocrates, Celsus, and Galen, show that venereal diseases were as common in their day as in ours.

The literature of China and Japan, especially that of the former country, contains very strong evidence of the prevalence of syphilis in



those countries from remote antiquity. A disease characterized by an initial lesion, and followed by phenomena referred to bones, skin, glands, and various viscera, and remedied by mercury, is described therein. Proksch has apparently demonstrated the existence of the disease among the ancient Egyptians.

The investigation of the early history of syphilis has furthermore been greatly hampered by its confusion with other diseases. Thus, it is known that it must have been early confounded with leprosy, if, indeed, many of the so-called cases of leprosy were not essentially syphilitic. Syphilis was likewise confounded with other diseases transmitted by promiscuous intercourse, by very many in these days being regarded as having a common origin with them.

Syphilis first attracted universal attention at the close of the fifteenth century, when the disease became epidemic throughout Europe. Two views as to the source of the epidemic prevailed. One was that the disease was imported from America by the sailors of Columbus; and the other that it was the awakening into activity of the germs of a disease which up to that time had escaped observation, because of its limited prevalence. Hyde's investigations seem to relieve Columbus of the odium of having imported the disease into Europe, for he shows the prevalence of an epidemic of the disease in Spain at the time Columbus sailed.

The past century has seen enormous strides in the advancement of our knowledge respecting the many phenomena of syphilis, advancements due to the labors of Ricord, Fournier, Hunter, Bell, Van Buren, Keyes, White, and many others.

Syphilis must now be regarded as a widespread disorder, one for which the clinician engaged in the investigation of chronic diseases must ever be on the alert. Esmarch has said that the disease is so widespread in Europe, that everyone may be considered as having acquired a partial immunity from the infection. No class of society is exempt from it. It is found alike among the occupants of the palace and of the hovel; and among the just and the unjust. Acquired generally in an act arising from moral weakness or deficiency of moral sense, it may also attack the innocent. Altogether too little attention has been paid to the disease by the physician in private practice. Too many times, indeed, he fails to see in the dignified and prosperous man of to-day the licentious youth of a couple of decades before.

As to the geographical limits of syphilis, it may be said that the prevalence of the disease in any community is in direct proportion to the importance of the commercial relations with the rest of the world. Still the disease may be found anywhere, for Park speaks of having seen cases of it among the natives of Central Africa. Even in one of the districts of South Africa it was stated that one-sixth of all the admissions to a certain hospital were for syphilis.

**Etiology.**—Syphilis arises from two causes, first, *inoculation*, and secondly, *inheritance*. As already intimated, the nature of the syphilitic poison is such as to require intimate contact of the infecting agent with an abraded surface or a delicate mucous membrane. In the vast majority of cases, the disease is transmitted by indiscriminate intercourse; though it may be transmitted accidentally in a variety of ways. Bulkley, of New York, has paid considerable attention to the study of syphilis arising from other than genital infection—*Syphilis insonitium*. His investigations show the possibility of the appearance of the initial lesion of syphilis in practically any portion of the body, though (aside from the genital organs), it is found in the majority of cases on the lips, the breasts and nipples, and in vaccination wounds. Physicians, especially surgeons and gynæcologists, run especial danger of inoculation in the pursuit of their duties. That this is not a chimerical fear is shown by the fact that in one large community, of which I have positive knowledge, 2 per cent. of the physicians, I know, have acquired syphilis in this way. There may be others similarly unfortunate whose cases have not attracted my attention. Physicians are sometimes responsible for the accidental infection of their patients by the use of improperly cleaned instruments that had previously been used on syphilitic patients. The great danger here is the use of instruments employed about the mouths of the syphilitic, as in dental work. It must be remembered that the syphilitic poison lodged on an instrument preserves its activity, though it be dried for a long time. The disease may be transmitted from one member of a family to another through the medium of towels, eating utensils, etc. Children sometimes acquire the disease through the carelessness of nurses who permit the little ones to be kissed and petted indiscriminately.

As to the virulence of the various syphilitic lesions and of the tissues and secretions of the syphilitic individual, there are great differences. In a general way it may be stated that all lesions of the early stage of the disease and their secretions may communicate the syphilis. The mucous patches about the mouth and the condylomata about the female genital organs are known to be especially dangerous. As to the virulence of the blood of syphilitic individuals, one cannot speak positively on all points. Inoculations performed with this fluid, taken from persons in the active stages of syphilis, have communicated the disease; it is not positively known, however, that infection may take place through the blood of persons in the latent stages of syphilis. As to the infectiousness of the physiological secretions, milk, semen, etc., opinions differ. The weight of evidence is against the communicability of syphilis by the milk, although Voss claims to have performed a successful inoculation with this fluid in one instance. As to the semen, some good authorities favor the view that it *may* be a medium of infection. Experimental inoculations with the semen have thus far failed to transmit syphilis,

and yet it has been claimed that a syphilitic man may communicate the disease to his wife by his semen, without first making her pregnant.

It is not likely that syphilis is communicable by pathological secretions, as pus, etc., from lesions other than syphilitic, unless they happen to be mixed with the patient's blood or the secretions of an essentially syphilitic character. It is well known that syphilis has been communicated by vaccination from humanized virus; but it is generally believed that had a pure lymph free from blood corpuscles been used, no infection would have occurred.

So far as the late lesions of syphilis are concerned, it is generally believed that they are non-infectious. Some few instances showing the contrary have been reported.

The active element in the infection of syphilis is believed to be a micro-organism, the exact nature of which cannot as yet be said to have been positively determined. All evidence bearing on the subject at present indicates that the bacillus of syphilis is a micro-organism, first described by Lustgarten. In size and shape it resembles closely the bacillus of tuberculosis, but differing from the latter in having knob-like swellings on the ends, and by the frequent appearance of bent and S-shaped forms. It is from two to seven-thousandths of a millimetre in length, and its width is about three ten-thousandths of a millimetre. It is contained in round cells, and is found almost invariably in the hard chancre, gummatous infiltrations and other syphilitic formations. It bears a close resemblance to the smegma bacillus, with which it has been claimed to be identical. Inasmuch as it is found in distant tissues and in the gummatous growths, this can hardly be the case.

As will be seen presently, syphilis is remarkable for its multiplicity of symptoms and its variations in intensity. The causes which lead up to these differences are believed to reside rather in the patient than in any special variations in the virulence of the infectious material. What these constitutional peculiarities are favoring a malignant course in one instance and an exceedingly mild one in another, are at present unknown. As a rule, weak, cachectic individuals suffer the most severely, and yet it is not unusual for a strong, robust person to be stricken with the disease in its most malignant type. Aside from the idiosyncrasies of the patient, there are certain factors of either local or general character serving to modify the intensity of the attack. Of these, climate, age, alcoholism, intercurrent maladies, diatheses, traumatism, and accidental infection with pyogenic microbes, are the most prominent. It is also believed that the dose of the poison has considerable to do with the severity of the disorder.

Syphilis exists in every clime. Its ravages are usually more moderate in temperate regions. The cutaneous manifestations are generally more severe in hot countries. Foreigners not yet acclimated to the region



in which they happen to dwell are apt to suffer more severely than do natives. Exposure to cold and dampness in the pursuit of ordinary occupations intensifies the lesions in the majority of cases.

While there can hardly be said to be any predisposition to severity of attack because of age, the fact that syphilis is apt to be more severe as it occurs in subjects at the extremes of life must not be overlooked.

Excessive alcoholic indulgence is undoubtedly a very important factor in shaping the course of syphilis. Tending as excesses in this direction do, to arterial and other degenerations, cutaneous troubles, etc., even apart from syphilitic infection, this tendency must become all the more active in the presence of such a constitutional poisoning.

The gouty and scrofulous diatheses undoubtedly exert a marked effect in determining the nature of the eruptions of the secondary stage of the disease.

Intercurrent maladies may exert one of several influences on the ordinary course of syphilis. Thus, the appearance of secondary manifestations may be hurried in one instance; in another (very exceptional cases by the way), the period of secondary eruption may be considerably modified or even made to disappear during the continuance of the intercurrent disease, only to reappear on its subsidence; and in still a third instance, of which an example may be found in variola, the syphilitic character impresses itself on the lesions of the intercurrent disease, which straightforth take on the features of a specific ulceration.

The relationship between traumatism and syphilis has a very practical side. The question is often asked in the case of a syphilitic subject about to undergo an important surgical operation, will his disease interfere with the proper healing of the operation wounds? Just so far as a debilitated cachectic constitution of any kind will interfere with proper convalescence after operation, so will syphilis exert its influence. So far as danger from suppurative complications is concerned, there need be no concern, if proper antiseptic precautions are adopted.

**Clinical Course.**—For purposes of convenience of description, the division of acquired syphilis into primary, secondary and tertiary stages is one of great convenience. At the same time it must be remembered that such a division is hardly correct, for from the moment of inoculation with the specific virus through the long term of months or even years, during which there appear in succession, the initial lesion, the adenopathy, the many cutaneous eruptions, and the great variety of visceral disturbances, there is one continuous pathological process, active at times, apparently dormant at others, but nevertheless always pursuing its way. For reasons not well understood, the tertiary manifestations often appear quite early in the course of syphilis; in others, they are postponed to an unusually late date; and in still others, fortunately the vast majority of all cases, they never occur.

It should be borne in mind that syphilis is by no means always a serious disorder. Many cases run a remarkably benign course, with symptoms so mild and transitory as to almost escape notice. Others, while presenting symptoms for the most part benign, so far as their intensity is concerned, exhibit a persistence of the same, or a remarkable tendency to relapses. Contrary to the usual conception of syphilis, it is just these benign types of the disease that constitute fully two-thirds of all cases. But there is another standpoint from which syphilis must be viewed. A certain percentage of cases is of a most malignant type. In all these cases the symptoms are profound and persistent, exhibiting oft-times a tendency to relapse, and even to extensive destruction of tissue. At no time in the course of the disorder is it possible to predict that of the future. Ordinarily, the case beginning benignly pursues that feature to the end. At the same time it must be remembered that the mild case in the beginning may become the most destructive in the end. It has even been said that it is the cases presenting mild secondary symptoms that develop the severe tertiary manifestations. In syphilis of the nervous system this seems to be the case, if personal experience is to be the guide, and this view is in accord with that of neurologists generally. Yet competent authorities deny that such a belief has any foundation in actual experience. Beginning now our consideration of the separate symptoms going to make up the clinical picture of syphilis, we come first to study the initial lesion of the disease, the so-called initial sclerosis, the hard chancre, or the primary sore.

**Initial Lesion ; Primary Syphilis.**—The initial lesion of syphilis is a sore appearing at the seat of entrance of the poison into the system. This, in conjunction with the secondary involvement of the lymphatic glands in closest anatomical relation to the affected part, constitutes what is ordinarily denominated the primary stage of syphilis. By some, it is believed that the initial lesion is infecting, but such are in a very small minority. Even before its appearance, the system must be regarded as infected. No better proof of this fact is necessary than the utter failure of all efforts to cure or prevent syphilis by the early excision of the primary sore.

As a rule, there intervenes a period of about three weeks between the time of inoculation and the initial outbreak. Still this period, which has been called the first incubation period, is liable to great variations. On the authority of Diday, it is claimed that it may last but twenty-four hours; and of Fournier, as long as seventy days. During this period of first incubation, any abrasion that may have existed heals completely, and no evidence of the terrible malady to come is discernible. If the parts are carefully studied, there first appears a small red spot, followed very shortly by a local induration and a papule. The surface of this is generally abraded, and exudes a scanty clear secretion. The

most characteristic feature of the lesion is the induration, which differs materially from the inflammatory thickening accompanying chancreoid. It is generally compared to a sensation as if a piece of parchment were beneath the surface. It is best appreciated when the lesion occurs in soft tissues, as the prepuce, while it is sometimes difficult of recognition in such resistant parts as the cervix uteri and the fingers. The initial sore disappears slowly, the induration oftentimes not going away entirely until after cutaneous symptoms have appeared. Sometimes a slight pigmentation of the surface remains and persists for some time. In the majority of instances there is but one sore. It sometimes happens, however, that the inoculation takes place simultaneously through several abrasions, in which case a lesion may mark each point of entrance into the system.

The syphilitic chancre may objectively present several types, of which we have the following: (1) The dry, scaling papule; (2) the superficial erosion; (3) the ulcerating initial lesion; (4) the mixed chancre.

The dry, scaling papule, as its name implies, consists of a simple papule surmounted by a slight desquamation. The abrasion is very superficial, indeed, well-nigh imperceptible to careless observation. It is usually observed on free surfaces, where it is not exposed to maceration of discharges. The "superficial erosion" presents a raw surface, the superficial layers of epithelium having been destroyed. It is apt to be covered with a gray film, especially at its centre. The "ulcerating lesion" is one which, from various causes, undergoes ulceration by reason of the low vitality of syphilitic lesions generally. The "mixed chancre" is one in which the primary inoculation has communicated both chancreoid and syphilis.

**ADENOPATHY.** Accompanying the syphilitic chancre is an affection of the anatomically associated glands. These are enlarged and indurated. This change is inflammatory in nature; but the pathological process is of a low grade, and the affected parts exhibit neither pain, redness nor heat. In the case of a genital chancre, a cluster of enlarged glands is observed in the groin, especially marked at the side of the initial sore. The glands along Poupart's ligament are the ones most commonly affected. They rarely undergo suppuration; in fact, such a process indicates infection by pyogenic micro-organisms.

**DIAGNOSIS OF PRIMARY SYPHILIS.** The essential diagnostic characteristics of the syphilitic chancre are its induration and the adenopathy above described. In cases of doubt, an examination of the patient from whom infection occurred, may be of some service. In others, one must have recourse to a careful study of the general clinical features of the "sore." The syphilitic chancre may occur from other exposure than venereal; the chancreoid is nearly always venereal in origin. The syph-



ilitic chancre arises from inoculation with the discharges of the initial and secondary lesions of syphilis; "chancroid" from the secretions of another "chancroid." The periods of incubation of the two sores are widely different. In syphilis it ranges from two to four weeks, as a rule; in "chancroid" it is hardly more than two or three days. Syphilis starts with a papule very often; chancroid as a pustule or ulceration. Syphilitic chancre is usually single, and not auto-inoculable; "chancroid" is frequently multiple, and is auto-inoculable. The borders of a "chancroid" are irregular in contra-distinction to the regular round or oval edges of the syphilitic sore. The glandular involvement of chancroid is usually limited to one gland, which commonly undergoes supuration.

**Secondary Syphilis.**—Following the initial lesion there ensues a period of second incubation, which varies in length, in different cases, from twelve days to six months, the average duration being about six weeks. Influences tending to malignancy in the course of the syphilis lessen its duration. Towards its close, that is, from two to four weeks before the advent of cutaneous manifestations, symptoms prodromic of secondary syphilis appear. These, as do all other symptoms of the disease, present the widest differences in intensity. In some cases they are entirely absent; in others so mild as to almost escape notice, and in still others so severe as to lead to the belief that some acute disorder is about to break out. They are never of such a nature as to point indubitably to the character of the disturbances about to appear. As a rule, they consist of a sallowness of countenance, tired or expressionless eyes, general feeling of discomfort, disturbances of sleep and digestion, loss of appetite, headache, etc. The latter symptom is often very prominent. It may affect any portion of the head. It is almost always greatly aggravated at night. Indeed, it may exhibit a remarkable periodicity, coming on at a stated hour each evening, disappearing with equal regularity each morning. This periodicity may even lead to a mistaken diagnosis of malaria. As in many infectious diseases, the spleen is somewhat enlarged. The liver is disturbed, and, in exceptional instances, jaundice appears. This latter symptom has been shown by Lancereaux to be the result of obstruction of the common bile-duct by the pressure of enlarged portal glands.

THE STATE OF THE BLOOD during this prodromic period and during the progress of the secondary symptoms affords an interesting study. It has been shown by most painstaking investigations that these partake of the essential characteristics of anæmia. Long before the appearance of the eruptions, even three weeks before, the number of red blood corpuscles is diminished, and the percentage of oxyhæmoglobin is lessened. These two changes exist in direct proportion to each other. At the same time the number of white blood corpuscles increases. All of these

changes are more marked during the different febrile exacerbations to be described very shortly.

During the stage of eruption all of these blood changes continue, but with increased severity, and especially are they marked when the eruptions are accompanied by fever. Each cutaneous outbreak increases the already existing blood changes. The blood returns to its normal state with the disappearance of the secondary symptoms.

**SYPHILITIC FEVER.** The ordinary conception of syphilis provides for a fever of high or low grade at the time of the secondary outbreak. While this is strictly true, it must also be borne in mind that fever may occur with every renewed cutaneous eruption throughout the course of the disease, the ulcerative and pustular lesions being always accompanied by a higher temperature than those of the papular and the erythematous varieties.

Ordinarily the fever occurring at the time of the eruption does not attain a high degree, ranging usually between  $101^{\circ}$  and  $102^{\circ}$  F. The evening temperature is generally about a degree higher than that of the morning. Exceptionally, however, hyperpyrexia ensues and the temperature ranges from  $104^{\circ}$  to  $106^{\circ}$  F. The severity of the fever seems to be determined in a measure by the same influences which tend to aggravation or amelioration of the other symptoms of syphilis, namely, personal idiosyncrasy and the general health standard of the patient.

**ENLARGEMENT OF THE LYMPHATIC GLANDS.** The lymphatic glands are found enlarged in certain localities in nearly every case of syphilis at the outbreak of the secondary stage. This glandular complication usually asserts itself during the period of secondary incubation, to reach its height with the full development of the eruptions. It is generally regarded as one of the earliest and most constant evidences of constitutional syphilis. While thus a symptom of diagnostic value, its importance must not be overestimated. Exceptional cases are observed in which it is entirely absent, while numerous instances occur in which healthy individuals have glandular enlargements in one or another portion of the body. In the case of constitutional syphilis, the glands most commonly affected are those about the neck, particularly the glands arranged along the posterior border of the sterno-cleido-mastoid muscle, and the jugular, supraclavicular, and axillary groups of lymphatics. The inguinal glands, which had hitherto escaped, participate in the changes. Much stress has been laid on the enlargement of the epitrochlear glands as pathognomonic of syphilis. While a valuable symptom, its importance has been overestimated, for it is absent in about one-fourth of the cases, and does not always indicate syphilis when present. Their discovery requires some little dexterity. The arm should be flexed at the elbow-joint. The fingers should be inserted gently into the sulcus, between the triceps and biceps muscles, at a

point two or three fingers' breadths above the internal condyle of the humerus.

The affected lymphatic glands range in size from that of a pea to that of a hazelnut. They are always painless, and never suppurate unless some extra-syphilitic infection occurs. In the latter case the suppurative process is apt to be more obstinate to treatment than in the case of adenitis of similar severity occurring in non-syphilitic individuals. The glandular enlargements of syphilis must be regarded, not as the result of sympathetic irritation, but as arising from the general toxæmia.

**SYPHILITIC CACHEXIA.** Syphilitic individuals, even before the secondary outbreak, oftentimes exhibit a cachectic appearance, which asserts itself with remissions or intermissions throughout the entire course of the disease.

**Cutaneous Symptoms of Syphilis.**—Occurring with a frequency greater than other symptoms of constitutional syphilis, the cutaneous manifestations of the disease form a very important part of its clinical course. Practically they are invariably present, so that their existence becomes almost essential to a diagnosis of syphilis. Keyes has shown indisputably that some few cases do escape the cutaneous ravages of syphilis. On the other hand, it must be remembered that these same symptoms might have been so mild as to escape attention, had they not been searched for, because of the warning given by the precedent chancre and its associated adenopathy.

The manifestations of cutaneous syphilis, it should be kept in mind, are capable of simulating closely any and all non-specific diseases of the skin. Hutchinson has put this fact aphoristically, that, "while syphilis may imitate all known forms of skin disease, it can create no originals." This being the case, how are we to recognize cutaneous syphilis when we encounter it? In the first place, we have the history of the case and the associated symptoms to guide us. In the second place, there are certain special characteristics of syphiloderms which are of no small diagnostic importance. They are as follows:

- (1) They develop slowly, and pursue a protracted course.
- (2) They are apt to develop in successive crops, the elementary lesions of which present the greatest variations.
- (3) The cutaneous lesions present are remarkable for their multiplicity of forms.
- (4) They are remarkable for their symmetry.
- (5) They tend to develop in curved lines or segments of circles, and arcs, and especially is this true of the papular syphilide.
- (6) Certain lesions exhibit a tendency to invade special portions of the body: the erythematous syphilide, the chest trunk, and flexor surfaces; the papular syphilide upon the face, brow, margin of the hairy scalp, back of the neck, trunk, and limbs; the squamous syphilide upon the palmar and plantar surfaces; the pustular syphilide upon the scalp



and hairy parts of the face; the ecthymatous and rupial syphilide, upon the lower limbs; and moist papules, at the natural orifices.

(7) Syphilitic eruptions present a distinctive color, which has been commonly described as a brownish-red or coppery tint, sometimes compared to the color of lean ham. When occurring in a dependent portion of the body, their tint may be of a bluish or purplish cast.

(8) The lesions disappearing, frequently leave behind them a pigmentation which, though not pathognomonic, possesses considerable diagnostic value.

(9) The scales of syphilitic eruptions are thinner, more superficial, less adherent, and less abundant than those of non-specific skin diseases. In color they are apt to be dirty grayish-white.

(10) The crusts of the pustular and ulcerative syphilides are characterized by their formation in superimposed layers, their unusual thickness, and their loose attachment to the lesions.

(11) The ulcerative lesions take on a horseshoe shape. This is due to the fact that healing takes place in the centre, while the spread of the ulceration proceeds by the convex margin. The edges of the ulcer are perpendicular and well defined, and its base is grayish or pseudo-membranous.

(12) The cicatrices are at first pigmented; later, they assume a clear, white tint.

(13) Pain and itching are remarkable for their absence in syphilides.

(14) The secondary cutaneous manifestations of syphilis are remarkable for their general distribution.

(15) Successive crops are liable to affect deeper and deeper portions of the skin.

**ALOPECIA.** Falling out of the hair is an exceedingly common symptom of secondary syphilis. It arises from two causes. In the first place it may be secondary to various eruptions of the hairy surfaces of the body resulting in disease or death of the hair-bulbs; or it may be due to the effect of the syphilitic toxæmia on the nutrition of these structures. The hair of any portion of the body may be affected, but attention is mostly directed to this trouble as attacking the scalp, beard, eyebrows, eyelashes, etc. It is an early symptom of secondary syphilis, occurring usually during the first year of the disease. Primary alopecia is always temporary. As already stated, the nutrition of the hair-bulbs is impaired; in consequence of this the hairs themselves lose their normal lustre, and become brittle and dry. The severity of the process presents the greatest variations. In some cases it is scarcely perceptible; in others it is marked over the entire scalp. When limited in distribution, it may affect any portion of the head.

Affecting the eyelashes and eyebrows it is characterized by the same irregularity, sometimes thinning out the entire parts, and sometimes occurring in patches.

In the consecutive or secondary syphilitic alopecia the loss of hair is confined to the seat of lesion.

Syphilitic alopecia is distinguished from that arising from other causes by the history of the case and by the distribution of the baldness. When the latter is premature or occurs from advancing years it affects mainly the scalp of the temples and the vertex.

**SYPHILITIC AFFECTIONS OF THE NAILS.** These are not mentioned in the present order because of their relative frequency, but by reason of their logical relationship to syphilitic affections of the hair, to which attention has just been directed. A study of syphilitic disorders of the nails shows them to be affected in a manner analagous to that of the hair. We have *onychia*, in which the matrices of the nails are diseased or their nutrition impaired, corresponding to primary alopecia; and *paronychia*, in which syphilitic disease invades the tissues about the matrices, and destroys them only by extension, corresponding to consecutive or secondary alopecia.

The nails may present quite a variety of changes in syphilitic onychia, though in no case are these diagnostic *per se*. The most common change is a thinning and cracking of the nail along its free border, a change strictly analagous to the increased brittleness and the dryness of the hair preceding alopecia. In other cases the nail becomes partially or wholly detached from the nail-bed, and is finally lost. A very rare variety of onychia is that known as the hypertrophic, in which the nail becomes enormously thickened. The free margin of the nail is thicker than the base, and the nail presents a whitish-yellow color.

Syphilitic onychia may invade either fingers or toes, and, of these, one or all. The trouble is characterized by its insidious onset and the very little pain attending it.

Paronychia may likewise involve the nails of the toes as well as those of the fingers. Three varieties have been recognized: (1) Dry syphilitic paronychia; (2) inflammatory syphilitic paronychia; (3) ulcerative paronychia. The first of these generally invades the fingers, and is characterized by thickening and exfoliation of the epidermis about the nails. The affected tissues become fissured and excoriated. The process may spread until the entire dorsal aspect of the terminal phalanx is diseased. This variety is especially liable to occur in persons of a gouty or rheumatic diathesis.

Inflammatory paronychia resembles very closely the ordinary "run-around," but differs from it in the absence of suppuration and its chronic course. In brief, it is a chronic inflammation affecting the tissues about the nails.

Ulcerative syphilitic paronychia is indicative of a bad type of syphilis. The ulceration encircles a part or all of the nail, and is surrounded by a dusky-red or violet congestion. The ulceration extends to the nail-

bed, and the nail is loosened and exfoliated. The entire terminal phalanx becomes infiltrated and clubbed. The discharge from the ulceration is profuse and offensive.

Syphilitic affections of the nails are observed generally during the first year of the disease, though their advent may be postponed until the eighteenth month. They are recognized by the history of the case, their association with the characteristic cutaneous symptoms of the disease, their chronic course, the absence of pain, and, aside from the ulcerative forms, of suppuration. As to the subsequent condition of the nail, much depends upon the extent to which the matrix has been affected.

**Syphilis of the Mouth and Throat.**—Occurring with equal frequency to that of lesions of the skin in secondary syphilis are the manifestations of the disease in the mouth and throat. In fact, the latter may be regarded as the cutaneous outbreaks appearing on the mucous membranes, modified in their clinical characteristics by reason of the heat and moisture and mechanical irritation to which they are subjected in this situation. Starting as erythema, papules, tubercles, pustules, etc., they speedily degenerate and form the mucous patches, the existence of which is so generally regarded as one of the necessary evidences of constitutional syphilis. Even the primary lesion occurring in this situation ultimately takes on this same tendency and becomes a mucous patch.

Syphilis of the mouth and throat is an early phenomenon of the disease. The lesions exhibit a remarkable tendency to recurrence. Their secretions possess exceedingly contagious properties, and, mingling with the saliva, make the latter especially dangerous. Ofttimes the resulting ulceration is very destructive, and yet, notwithstanding the great loss of tissue that sometimes ensues, the resulting cicatrix is disproportionately small. Local causes, other than the natural heat and moisture and the mechanical irritation of food, favor their development, *e. g.*, improper care of the mouth and teeth, the presence of decayed teeth and pyogenic affections of the gums. The tongue and palate suffer, the ulceration sometimes even perforating these structures.

**Syphilis of the Eyes.**—Any of the structures of the eye may be the seat of syphilitic lesions, notably however, the iris, the cornea, the optic nerve, the retina, and the choroid. The objective and subjective symptoms of these different conditions will be found in any text-book on ophthalmology and need not be repeated at this time. The most frequent lesion observed is an iritis, which usually appears during the secondary stage of the disease. Interstitial keratitis is very common in hereditary syphilis.

**Syphilis of the Joints, Muscles, etc.**—Early in the disease, sometimes even before the eruptions appear, the syphilitic patient is tormented with pains in the joints. In many instances these are the result of a purely functional affection—an arthralgia, the subjective symptoms alone



being present. In other cases the synovial membranes are invaded ; in still others, disease of the cartilages and even of the bones, lies at the foundation of the difficulty. Many of the rheumatic pains of secondary syphilis are due to affection of the muscles, the pathological change being one of inflammation—a myositis, in some cases of an irritative variety, and in others interstitial.

Recapitulating then, we find secondary syphilis characterized by a group of phenomena consisting of a multiplicity of cutaneous lesions, glandular enlargements, mucous patches in the mouth and throat, chloro-anæmia, fever, iritis and alopecia. Exceptionally the secondary stage is characterized by disturbances of the nervous system, and other phenomena which one is accustomed to expect only in the tertiary stage.

**Tertiary Syphilis.**—OF THE SKIN. It must be remembered at the outset that the majority of syphilitic individuals escape the tertiary ravages of the disease entirely, either by reason of good treatment in the early days of the disease, or because personal predisposition does not favor such a dire result. The tertiary lesions of the skin differ from those of the secondary stage in the first place in their local distribution. They exhibit no tendency whatever to symmetry. They are deep-seated, and are usually destructive. They develop without any regularity, and show a tendency to spread locally. They are non-contagious.

Affecting the tongue, mucous membranes, etc., the usual lesion is at first a gumma, which is either absorbed as the result of treatment, or undergoes degeneration and ulcerates.

The bones are affected in the tertiary stage. The lesions here may proceed to caries or necrosis. Especially are the bones of the nose liable to disease, giving rise to many serious phases of ozæna.

It is during the tertiary stage of syphilis that we observe the many involvements of the various viscera and the central nervous system, either by reason of the syphilis attacking these structures directly or through the medium of different types of vascular disease.

### SYPHILIS OF THE LARYNX.

Syphilitic disease of the larynx is undoubtedly of common occurrence. Many cases escape recognition, because of failure to make laryngoscopic examinations, the knowledge that the patient is suffering from syphilitic infection being deemed sufficient information for therapeutic purposes. Probably few cases escape some laryngeal complications in the course of one of the stages of the disease. The same causes that excite acute and chronic catarrhal laryngitis, namely, exposure to cold and foul atmospheres, and indulgences in alcohol and other excesses, predispose to the localization of syphilis in the larynx.

Laryngeal syphilitic lesions may be divided into those of the secondary and tertiary stages respectively. In the secondary stage of syphi-

ilis the lesions affect mainly the mucous membranes and the submucous tissues. The tissues present an appearance resembling very closely that observed in ordinary catarrhal inflammation. Differentiation is often possible later, when the parts manifest a tendency to multiple superficial ulcerations. Sometimes these ulcers coalesce, and form one large ulcerating surface. There is really nothing characteristic in any of these lesions. Mucous patches have been described. They must be very rare. Their existence has been strenuously denied by some excellent authorities.

The lesions of the tertiary period are gummata, fibroid degeneration, and tertiary ulcerations. *Gummata* may be single or multiple, and of various sizes and shapes. They are usually found in the submucous tissue of the free border and posterior surface of the epiglottis and in the interarytenoid space. The mucous membrane by which they are covered is intensely red. As they increase in size, however, this membrane thins out, and displays the yellow or whitish-yellow color of the tumor beneath. As the pressure increases, the nutrition of the membrane suffers, and necrosis takes place.

FIBROID DEGENERATION is a condition observed only in cases of neglected syphilis. It consists of a remarkable tendency to the formation of fibroid tissue within the laryngeal structure. The contraction of this tissue leads to narrowing of the lumen of the larynx, and even to the conversion of the organ into a dense cicatricial tube. In some cases the formation of the fibroid tissues is such as to develop tumors, which have been mistaken for gummata. They differ from the latter growths, however, in their pale grayish or whitish appearance. Ulceration sometimes accompanies this condition, and may prove to be a condition of great danger. The course of fibroid degeneration is steadily progressive.

TERTIARY ULCERATIONS are more or less circular in their outlines, and have a grayish or lardaceous base. Especially characteristic is the zone of intense inflammation by which they are surrounded. Their discharge is very offensive, and of dirty yellow color. The favorite site of these ulcers is the free edge and lingual surface of the epiglottis. The cicatrization following the healing produces marked deformities. The epiglottis may be distorted or bound in different positions by cicatricial bands. Sometimes the ulceration being unchecked, attacks the cartilages, which may slough. This invasion of the cartilages is always a serious matter, for not only does their destruction involve impairment of the laryngeal functions, but their death may be productive of general infection and other accidents. The tendency to acute oedema in these cases must always be borne in mind. Cicatricial deformities of great variety follow these laryngeal ulcerations.

The greatest diagnostic difficulty is encountered in the differentiation of syphilitic and tuberculous ulceration. This difficulty is especially

great in cases in which tuberculosis is engrafted on a syphilitic constitution. The appearance of but one ulcer of rapid development is in favor of syphilis. Tuberculous ulcerations are apt to be multiple, and are slow in development. Syphilitic ulcerations are larger than the tubercular, and attack by preference the site already named. Tuberculous ulcerations are especially prone to invade the lower and posterior portion of the larynx. The syphilitic ulcer is surrounded by a deep-red inflammatory zone. The secretion of the tuberculous ulcer is quite profuse and gives the breath a peculiar sweetish odor; syphilitic ulceration has a less profuse secretion, which is highly foetid. Laryngeal syphilis presents but little pain as a rule. In laryngeal tuberculosis, pain is intense.

*Cancer*, according to Cohen, exhibits a remarkable tendency to attack syphilitic individuals, thus making a differentiation of cancerous and syphilitic ulcerations a very important matter. Cancer is prone to occur after middle life, and is of much less rapid development than syphilis. The tumor generally produces some œsophageal obstruction before interfering with the functions of the larynx. Hæmorrhages are apt to be profuse. The secretions from the mass when it undergoes ulceration, are in no wise different from those of malignant ulcerations elsewhere.

**Prognosis.**—In the laryngeal lesions of secondary syphilis the prognosis is good, as it is also in the majority of the cases occurring in the tertiary stage, especially if they come under treatment early. Fibroid degeneration is steadily progressive.

**The Treatment** consists of the application of cleansing sprays, and the subsequent use of such local remedies as iodo-glycerin, etc. The constitutional treatment is of the greatest importance, and consists of the administration of mercury in one of its numerous forms during the secondary stage, and of iodide of potassium in the tertiary stage. *Aurum*, *graphites*, *mezereum*, and *nitric acid*, but especially the first two, are often useful. The reader is referred to the special section on syphilis for further suggestions as to treatment.

## SYPHILIS OF THE NERVOUS SYSTEM.

This subject may best be studied first by a general survey of the field, and then a special consideration of the way in which syphilis affects the brain, the spinal cord and the peripheral nervous system, respectively. In the making of a diagnosis in any given case, we have, of course, the clinical history as a guide. But this not always forthcoming, and one must rely solely upon his clinical foresight for a decision. There is hardly a datum of more value than the age of the patient. With the exception of certain diseases possessing a well-defined symptomatology, and peculiar to early life, *e. g.*, Friedrich's disease, epilepsy, poliomyelitis anterior, etc., fully 80 per cent. of all organic diseases of the nervous system occurring prior to the fiftieth year of life are syphi-



litic in origin, providing, of course, other ordinary causes of organic nervous disease in young adults can be excluded. Especially does this remark apply to hemiplegias and epileptiform seizures, occurring for the first time between the ages of twenty and fifty. The non-syphilitic cases include cases of embolism, cerebral changes secondary to renal disease, chronic alcoholism, abscesses, traumatic affections and malignant growths. The importance of age can be understood in the case of cerebral paralysis dependent upon thrombosis. Aside from acute pyæmia, thrombosis occurs only in connection with endarteritis and atheroma. Now atheroma is limited to the aged; endarteritis occurs in the young in connection with syphilis. In either case it is plain that the symptoms must be exactly the same, for they result from the deprivation of certain areas of the brain of blood. If these symptoms occur in the young and we are able to exclude renal disease and chronic alcoholism, syphilis is almost certain.

A difficulty will be occasionally met with in cases in which we find, not only a syphilitic history, but also evidences of chronic organic heart disease. Here we must call to our aid the associated conditions. Evidence of the activity of the syphilis or the heart lesions must be sought. Thus if the endocarditis is of recent origin, and there are indications of embolism in other organs than the brain, we must ascribe the attack to that cause; but if the paralysis has been preceded by some headache, transient spells of numbness, fits of somnolence, etc., we can rest assured that there is syphilitic disease of the arteries.

Taking up *seriatim*, the various symptoms common in syphilitic cerebral disease, I shall first refer to the headache. This is by all odds the one most frequently present. It varies in character, position and intensity in different cases. It often exists for years before other symptoms assert themselves, and not infrequently disappears on the supervention of paralysis, convulsions and other phenomena. Its only characteristic feature is its nocturnal exacerbation, which, however, is not to be noted invariably. While usually persistent it is often paroxysmal, or has periods of maximum and minimum intensity. The involvement of the pericranium or the dura mater in the pathological process leads to tenderness over the painful areas. In such cases, the headache itself is apt to be of unusual severity. Neuralgia of the fifth pair of cranial nerves as a result of syphilis is never an essential neuralgia, that is, an affection dependent upon functional disease of the fifth pair of nerves. When neuralgia of the fifth does result from syphilis, it is indicative of a meningeal inflammation or exudation at the roots of these nerves, or of inflammation or pressure in their course.

Epileptic attacks are of frequent occurrence in connection with cerebral syphilis. The characteristics of the seizures will, in many cases, seem to be indistinguishable from those of genuine epilepsy at

first sight. A little study of the phenomena will soon discover points of difference. Usually the character of the convulsion is that of organic disease of the brain or its membranes, *i. e.*, meningitis, tumor, etc. The seizures are thus apt to be of long duration, that is, half an hour or upwards; and they are associated with severe headaches, with nocturnal exacerbations, remarkable exaggeration of the knee-jerks, etc. In idiopathic epilepsy the convulsions rarely, if ever, last more than fifteen minutes; in the vast majority of cases not more than five; they are not associated with headache in the inter-paroxysmal periods, and the knee-jerks are normal. Of equal diagnostic value with the above is the age of the patient. A large majority of convulsions of epileptiform type coming on for the first time after the twenty-fifth year of life may be regarded as of syphilitic origin, providing renal disease and traumatism can be excluded. It will sometimes be noticed that the convulsions of syphilitic epilepsy are associated with more or less paralysis. While the loss of power is usually limited to the areas that have been convulsed, this is by no means invariably the case. In some instances the paralysis is the first symptom, the convulsions occurring later. Sometimes the convulsions are purely of the Jacksonian type. Exceptionally we meet with cases of syphilitic epilepsy the symptomatology of which differs in no wise from that of the idiopathic disorder. Then we have only the age of the patient and the venereal history to guide us.

The paralyses of cerebral syphilis are remarkable for their variety, and are characteristic of the time of life at which they occur. Their variety, both as regards their distribution and modes of onset, may well be understood when one recalls the numerous pathological conditions that may occur in the cranial cavity as a result of syphilis. The disease may involve the skull, the meninges, the cerebral vessels, or the brain itself. It may give rise to syphilitic nodes, gummata, endarteritis obliterans, cerebritis, neuritis, meningitis, periarteritis, aneurism, apoplexy, or acute softening. Syphilitic palsies may thus come on suddenly or slowly. Their distribution depends largely upon the area of brain involved. There is one feature that is markedly characteristic of them, and that is their incompleteness. Motor function of the paralyzed part is not entirely destroyed. The paralysis in such cases is temporary. Of course, in those instances in which the palsy has arisen from syphilitic diseases of the bloodvessels, and rupture of the same, its characters must be the same as when it results from vascular disease consequent upon the ordinary degenerative processes of advancing years. In such cases they will in all probability be complete. There is a form of hemiplegia especially characteristic of syphilis, and that is one consisting of numerous slight attacks with or without loss of consciousness, coming on suddenly and disappearing in a very short space of time.

Another remarkable characteristic of syphilitic palsies is the fre-

quency with which they involve certain cranial nerves, especially those giving motor power to the ocular muscles. So rarely, indeed, does motor-oculi palsy occur in adults in conjunction with other causes than syphilis, that I feel safe in saying that no amount of confidence in a patient's veracity or virtue should permit the physician to diagnose anything but cerebral syphilis in case of hemiplegia, or convulsions in which paralysis of one or more of the oculo-motor nerves is present. In some cases, indeed, the oculo-motor paralysis may be the only evidence of intracranial disease.

Gowers describes a form of motor-oculi palsy characterized by its special tendency to relapse or persist. One third nerve becomes paralyzed, and improves to a certain extent under treatment, when the other becomes involved, and at the same time the one first attacked relapses. The nature of these cases is regarded as uncertain. The course and limitation of the symptoms do not point to nuclear disease.

The frequency with which the ocular nerves are affected in syphilitic brain diseases is due to the long course which they take at the base of the skull before entering the orbit, in contact with the meninges, and over a bony floor, in a region where syphilitic lesions are most commonly situated. The paralysis may affect an entire nerve, or but one of its branches. It may be limited to the pupil. They are often but premonitory of other symptoms.

While the ocular nerves are the cranial nerves most frequently affected in cerebral syphilis, the others do not escape. I have seen cases in which a very large number of these nerves were involved, notably one, in which every cranial nerve from the first to the eighth inclusive, was affected.

Althaus has laid claim to excessive exaggeration of the knee-jerks as pathognomonic of cerebral syphilis. In a general way it may be stated that syphilitic brain disease is prone to association with this, symptom. But so are most intracranial difficulties, though in less degree. Excessive tendon reactions must be regarded as suggestive, but by no means as pathognomonic.

A form of coma peculiar to syphilis has been described by Althaus. Cases of this character are rare, still their associations are such as to make their exact nature certain. The premonitory symptoms of syphilitic coma are headache, mental confusion, drowsiness, indistinctness of speech, numbness and some slight loss of power. The symptoms of the attack set in during sleep, the patient being found in bed in the morning apparently unconscious. In some cases in which the coma is not profound, the patient may be aroused sufficiently to answer a question, but he immediately relapses into his former condition. The tongue is dry and is protruded with difficulty on command; swallowing is not readily performed. The eyeballs are sunken in their sockets, and there is appar-



ently a slight outward deviation of the eye. The greater the coma, the greater is this outward deviation. Reflex excitability everywhere is diminished. The muscles throughout the body are in a condition of great relaxation. There is incontinence of urine and fæces. The pulse is usually quite slow, but its character is variable. Respiration is slow and shallow, and the temperature is below the normal. Syphilitic coma is to be distinguished from ingravescient apoplexy by the age of the patient; apoplexy occurring after the fiftieth year of life, and syphilitic coma before that time. Opium poisoning and hæmorrhage into the pons, both having contraction of the pupils and coma, may be confounded with the syphilitic affection under consideration. In both of these conditions, the myosis is extreme, that is to the extreme limit possible, which is not the case in syphilitic coma. In opium poisoning fæces and urine are both retained. The coma of epilepsy is of shorter duration than the syphilitic variety, and should occasion no difficulty in diagnosis. In alcoholic coma the breath of the patient settles the question and the pupils are large. Uræmic coma is to be distinguished by the previous history, the albuminuria, the low specific gravity of the urine, uræmic headache and vomiting, and the complication of convulsions.

Aphasia is a frequent symptom of syphilitic cerebral disease. Coming thus it presents no special characteristics to distinguish it from the non-specific aphasia, other than those peculiar to syphilitic cerebral affections in general. Owing to the liability of syphilitic cerebral lesions to multiplicity, the aphasia is not infrequently associated with symptoms unexplainable on the supposition of a single lesion anywhere near the speech centres. Thus it has been observed that left hemiplegia occurs with comparative frequency with aphasia of syphilitic origin. This has led Wood to suggest that aphasia with left hemiplegia is almost certainly syphilitic.

A cerebral paralysis following a spinal lesion with a period of health intervening, may be regarded as almost certainly syphilitic.

Syphilis not infrequently gives rise to mental impairment. Acute mania and acute meningitis have been observed in the secondary stage of the disease. In the later stages we meet with transitory dementia, general paralysis of the insane and other insanities. Concerning the relationship between syphilis and general paralysis, it seems to be pretty well settled by most observers, that the latter disease occurs with too unusual frequency among syphilitic individuals to be a mere coincidence. It has been argued that the differentiation of certain cases of cerebral syphilis and general paralysis is impossible. Syphilis undoubtedly is capable of producing a meningo-encephalitis, and the lesion of general paralysis is a chronic meningitis with subsequent atrophy of the brain. General paralysis frequently precedes or follows locomotor ataxia. This

latter disease is now generally regarded as syphilitic in the vast majority of instances, hence one is justified in assigning paralytic dementia the same cause.

**SYPHILIS OF THE SPINAL CORD.** The symptomatology and pathology of spinal syphilis are not well understood. Cases of this character are as yet supposed to be infrequent as compared with cases of cerebral syphilis, and post-mortem examinations are rarely obtained, for the majority of these cases make a good recovery. As for data in framing a diagnosis, I think the same principles prevail here as in the case of cerebral affections: the age of the patient, the apparent multiplicity of the lesions, the lightness of the symptoms in proportion to their widespread distribution, and the exclusion of typical non-syphilitic diseases. As the syphilitic process may attack either the vertebræ, the meninges or the cord itself, one can readily understand the great symptomatic differences that spinal syphilis must present. Ofttimes, indeed, cases may show a most confusing resemblance to any of the typical system diseases of the cord.

It has been claimed that spinal syphilis is primarily the result of vascular disease, a view that is not confirmed by pathological investigations. Rather it would seem to be the result of a specific infiltration of the meninges, or possibly of meningo-myelitis. This process may attack any portion of the cord. It exhibits a tendency, however, to localize itself along the lateral columns, exceptionally to involve the posterior columns and the anterior horns. This tendency to attack certain fibre-systems of the cord renders these cases liable to closely simulate the idiopathic degenerations occurring in these regions.

Localized pain seems to be a valuable means of differentiating the syphilitic from the non-specific affections. In the former, as already stated, the meninges are affected. Some irritation of the posterior nerve roots thus ensues. The pain may be fixed in one spot of the spinal column, or it may appear in different portions of the body and be lightning-like in character.

Erb has described a type of syphilitic spinal disease now known as Erb's type of syphilitic spinal paralysis. The patients present the walk, posture and motion of spastic paralysis with greatly exaggerated tendon reactions, but with comparatively slight muscular tension. The sphincters are always affected. Sensation is more or less impaired, though usually but slightly so. The development of the disease is slow and improvement ensues on energetic anti-syphilitic treatment.

The etiological relationship between syphilis and locomotor ataxia will be considered in another portion of this work, that relating to the etiology of locomotor ataxia. Let it be here understood that the writer firmly believes that such a relation has been thoroughly established. There is a class of spinal cases preceded by well-marked ocular palsies differing only from tabes dorsalis in the marked prominence of this

symptom as an early phenomenon. Such cases seem to do better under anti-syphilitic treatment than do the typical cases of tabes, and now are beginning to be recognized as cases of spinal syphilis rather than of tabes dorsalis.

**SYPHILIS OF THE PERIPHERAL NERVOUS SYSTEM.** Syphilitic disease of the peripheral nerves is probably of rare occurrence. Most of these cases depend upon syphilitic disease starting in surrounding tissues.

**HEREDITARY SYPHILIS** is not commonly recognized as a cause of nervous disease—unjustly so we believe. As thoroughness in clinical study increases, the frequency with which hereditary syphilis of the nervous system will be recognized will be greater. It is a general belief among the laity—a belief that is becoming more common among the profession—that hereditary syphilis is the cause of many cases of idiocy, imbecility and epilepsy. Those who have had the most experience in the observation of idiots and imbeciles antagonize this view. Langdon Down states that not more than 2 per cent. of his cases were thus occasioned. Mr. Jonathan Hutchinson, whose experience in syphilitic affections is a very large one, considers that idiocy and hereditary syphilis are rarely associated. Statistics of large institutions for the feeble-minded confirm the conclusions of Down and Hutchinson. Shuttleworth, on the other hand, takes the ground that congenital syphilis occasions idiocy far more frequently than it receives credit for doing. The arguments advanced by him are that parents avoid the giving of a syphilitic history when entering their children into the asylum, and mental deterioration is not always evident until the period of second dentition, at which time it is often accompanied by paralysis, convulsions, and other degenerative symptoms, thus throwing the cases out of the class of idiopathic imbeciles or idiots.

Hereditary syphilis can, and often does, produce disease of the bones, those of the cranium not being excepted. The various forms of osteitis may readily lead to microcephalus and other conditions resulting in secondary cerebral changes. Inflammation of the different membranes of the brain has been observed in children of syphilitic inheritance.

It is extremely doubtful if idiopathic epilepsy is ever the result of congenital syphilis. Gowers, in his investigations of over one thousand cases of epilepsy, obtained a syphilitic history in only four cases. This percentage seems extraordinarily small, and the result of inaccurate information, for one would expect to find a greater percentage of syphilitic histories in a thousand so-called healthy families taken at random. This is true in this country at least, and London can hardly be said to be an improvement on American morals. When congenital syphilis does give rise to convulsions in childhood or infancy, these convulsions are very apt to be symptomatic of organic disease of the brain, and not of true epilepsy. Shortly after graduation, I attended a boy residing in



the slums, concerning whose parents no information was obtainable, and who was suffering from interstitial keratitis. He was also subject to convulsions, which recurred at intervals of one to two weeks. Under treatment the keratitis disappeared. Bromide of ammonium given in the 1x trituration by advice of Dr. James Kitchen, lessened the frequency of the convulsions most decidedly. As soon as the boy was able to go out, my attendance ceased. Six years later he applied for treatment at the Hahnemann College Dispensary of a most malignant ulceration of the face. Not long after I had ceased treating him, he developed a hydrocephalus, from which he recovered. The convulsions had also disappeared.

Hydrocephalus is sometimes the result of congenital syphilis. Sandoz, after a thorough study of the subject, basing his conclusions on four cases observed by himself and several others, says: "(1) There is a distinct syphilitic form of hydrocephalus. (2) It is congenital, or shows itself during the first month of life. (3) It probably arises from some inflammatory process of syphilitic origin in the ependyma of the cerebral bloodvessels."

Cases bearing a strong resemblance to tubercular meningitis, it is now generally admitted, may arise as a result of congenital syphilis. It is claimed that nearly all recoveries from tubercular meningitis are really cases of recovery from congenital syphilitic brain disease.

All cases of brain disease arising from congenital syphilis do not present as simple a clinical picture as epileptiform convulsions, hydrocephalus, idiocy, etc. Thus Dr. Angel Money exhibited before the London Pathological Society specimens from a case of chronic syphilitic meningitis, arteritis, cerebral atrophy and sclerosis in a boy aged four years. This patient was first seen at the age of eleven months, at which time he suffered from hydrocephalus. His skull was natiform, the root of the nose depressed, and there were signs of syphilitic disseminated choroiditis. The syphilitic taint was present in every member of the family. Under treatment the boy improved very much, and seemed almost well, when a left spastic hemiplegia developed. Two or three months later he lost his speech, developed right spastic hemiplegia, and one week later, died. At the autopsy, the brain and dura were found adherent everywhere, but the adhesions could be easily separated. The arteries were much diseased, as was also the brain substance.

As further illustrative of phases of cerebral syphilis in childhood, I may refer to two cases occurring in the same family, and reported by Warner. A syphilitic history was readily obtainable in both. Case I was that of a boy aged seven years, who suffered from severe pains in the head, which came in paroxysms, associated with vomiting. These attacks became more and more frequent, and were attended by loss of control over the bladder. Mercurial inunction failed to cure. He died four years later without the intervention of other symptoms. Case II

was well as an infant. At the age of six months he had bilateral convulsions, which continued until he was six years of age, when they ceased. At nine years of age he became clumsy and dropped things easily. At the age of eight he had a severe illness, and for six months was said to have meningitis. There was temporary squint and severe head-pain, with short-lasting powerlessness. One year later there appeared complete left hemiplegia, with rigidity and athetoid movements of the fingers; the left side of the face became partially paralyzed. There were traces of an old optic neuritis. The left knee-jerk was exaggerated; the right was normal.

Notwithstanding Hutchinson takes a strong stand against the frequency of diseases of the nervous system as a result of congenital syphilis, we find his book teeming with most interesting examples of this condition, to which I may here make slight reference with advantage to my readers.

A young woman had had numerous symptoms of hereditary syphilis. At the age of nineteen she had convulsions, which left her with right hemiplegia, which was passing away at the time the examination was made.

A girl, aged eleven, was liable to severe headache and epilepsy. She was deaf, and had the characteristic teeth and physiognomy.

A lad, aged nineteen, became liable to epilepsy at eleven, the attacks being attended by spasms in the muscles of the left limbs. These spasms preceded loss of consciousness; indeed, the latter was sometimes lacking.

A girl, aged fourteen, brought before the London Pathological Society by Mr. Nettleship, and who had paralysis of the third, fourth, fifth and sixth cranial nerves, the condition having existed for four years. The anæsthesia was complete, as was also the paralysis of several of the recti.

A case of fusiform neuritis in connection with inherited syphilis is reported by Ormerod, in a young woman of twenty-three. There was a fusiform enlargement of the median nerve in the middle of the upper arm. The tumor was tender on pressure, and was associated with motor paralysis, wasting and anæsthesia.

The age at which nervous symptoms may develop as a result of congenital syphilis has been variously stated. It is certain that they may appear shortly after birth, and that they may be delayed until early adult life. Whether they may occur at the age of maturity, without having been preceded by other syphilitic manifestations, must still remain *sub judice*. Other syphilitic phenomena may thus be delayed, and we know of no reason why the same should not apply to hereditary syphilis of the nervous system.

Hereditary syphilis seems to bear but little relation to disease of the

spinal cord. The only affections to which it may give rise in this portion of the body, seems to be paraplegia from syphilitic caries of the vertebræ.

### SYPHILIS OF THE HEART AND THE BLOODVESSELS.

Syphilitic disease may attack the heart or its membranes. The pericardium itself is involved only with the greatest rarity. Gummatous masses and inflammation have been reported as attacking this structure.

The heart itself may be the subject of quite a variety of direct and indirect syphilitic lesions. Thus there may occur gummatous growths and various changes due to the syphilitic blood and vascular changes. Cardiac gummata may attack any portion of the organ, though according to Lanceraux they are most frequently observed in the walls of the left ventricle, and to Councilman, in the interventricular septum. Though usually small, they may attain considerable size. In congenital syphilis they sometimes assume the miliary type. In many cases the growth disappears, the affected tissues undergoing a fibroid change. This latter condition leads naturally to a local weakening of the walls, and cardiac aneurism results.

Syphilitic fibro-myocarditis sometimes occurs. This condition is usually consequent upon vascular changes, and differs pathologically in no wise from the fibro-myocarditis arising from arterio-sclerosis. The very frequent association of syphilis with alcoholic excesses necessarily makes the part played by the former in the production of the arterial changes to a certain extent problematical.

The etiological relation between syphilis and aneurism of the aorta and general arterial disease will be fully considered in the chapters treating of aneurism and arterial diseases generally.

Symptoms arising from syphilitic diseases of the heart and arteries possess few if any characteristic clinical features. Rosenfeld has described a peculiar form of dyspnœa, which he attributes to syphilitic heart disease, and which he has designated syphilitic asthma. The symptoms come on when the patient is at rest, even at night, arousing him from sleep. Sudden death is believed to be the most common termination of syphilitic heart disease.

### SYPHILIS OF THE LUNGS.

Syphilis of the lungs may result from either the congenital or acquired form of the disease. As a sequel of acquired syphilis, it is very rare. The usually accepted belief in the common occurrence of a syphilitic phthisis is not borne out by carefully conducted autopsies, most of such cases being shown, beyond doubt, to be instances of tuberculosis in syphilitic subjects, while the balance of the cases may be considered as probably of the same nature. One of the facts on which the syphi-



litic nature of certain cases is based, is their improvement under iodide of potassium. Now, there is no fact in the therapeutics of phthisis better established than this, that the local process improves under any measures tending to elevate the general constitutional standard. The potassium iodide does this in syphilitic individuals, and so improves the pulmonary condition, tubercular in origin though it be.

The syphilitic lung-lesions may be enumerated as follows: (1) White pneumonia of the fœtus. (2) Gummata of the lungs. (3) Fibrous interstitial pneumonia. (4) Ulcerative lesions.

(1) **WHITE PNEUMONIA OF THE FŒTUS.** This was first described by Virchow as white hepatization, and its relation to syphilis established by Hecker. The pneumonic process is widespread, involving whole lobes, or even the entire lung. The consistence of the diseased portions is firmer than normal, and presents a grayish-white color on section. The alveolar walls are greatly thickened and infiltrated, and the alveoli themselves are filled with large, swollen, fatty, epithelial cells. The clinical importance of this disease is very small, as the subjects of it are either still-born, or expire within a few days after birth.

(2) **GUMMATA OF THE LUNG.** These are usually the result of acquired syphilis. They may invade any portion of the lung. According to Councilman, they are observed most frequently in the lower or middle and posterior portions of the lungs; according to Osler, they are most numerous towards the root. They vary greatly in size, some attaining a bulk equal to that of a goose-egg. In some respects they resemble tubercular lesions, but present important distinctions, which are all sufficient for differentiation. While the tendency in both is to cell-degeneration and caseation, this process is slower in gummata, and is preceded in them by a connective tissue development. The caseous mass in gummata is firmer than in tubercle. Fresh gummata have an opaque, grayish, or grayish-red color, and the lung surrounding them is more or less consolidated. The older gummata are opaque white, and are surrounded by a layer of dense, pearly, transparent connective tissue. Tubercular lesions, serving as foci of infection, are surrounded by new tubercular formations.

(3) **FIBROUS INTERSTITIAL PNEUMONIA.** This is characterized by a general and diffused formation of connective tissue. The alveolar walls atrophy and the capillaries become obliterated. It may be accompanied by the formation of gummata.

The abnormal formation of connective tissue may develop along the line of the bronchi and the bloodvessels extending towards the pleura. The result is more or less destruction of lung-tissue by the substitution of the connective tissue, and also by the compression exerted by the cicatricial contraction of the latter. Considerable deformity of the lungs results. The pleural surfaces show numerous depressions. The parts

between are emphysematous. The calibre of the bronchial tubes becomes greatly altered, being narrow in places, dilated in others.

(4) **ULCERATIVE LESIONS.** These are exceedingly rare, and find their origin in the breaking down of gummata.

The diagnosis of lung syphilis is a very difficult matter, even in the presence of a confessed syphilitic history. The symptomatology closely resembles that of tubercular troubles. Fever is usually absent and dyspnoea is more prominent than the physical signs would seem to indicate. Physical examination locates the lesion in a different portion of the chest from that in which one expects to find the lesions of tuberculosis. The cough is usually hard and dry and the sputum grayish and transparent. Microscopical examination fails to find the tubercle bacilli.

### SYPHILIS OF THE DIGESTIVE TRACT.

The mouth and the rectum are the portions of the digestive tract most commonly affected by syphilis. The former has already been considered. Syphilis of the œsophagus, stomach and intestines is very rare indeed. Ulceration of the œsophagus has been reported, both as an initial lesion, and as an extension from the pharynx.

The disease seems to attack the stomach only in the shape of gummatus infiltration of its mucous membrane, with or without ulceration. The same is true of syphilis of the intestines. Syphilis of the œsophagus, stomach and intestines possesses but little practical importance, as it is exceedingly rare and is not accompanied by symptoms sufficiently characteristic to make its diagnosis at all probable.

### SYPHILIS OF THE RECTUM AND ANUS.

Here syphilitic lesions are frequent in all stages of the disorder. Erosions about the anus, following erythematous or other rashes in this location, are quite common in the earliest days of secondary syphilis. Appearing almost as early in this same locality are the mucous patches. These have been divided into several varieties, as follows: (1) The small red papules. (2) Fissure-like cracks. (3) The small round gray patches. (4) The "plaque porcelanique." (5) The elevated or vegetating patches. These names are so far self-explanatory as to require no definition other than the fourth, which, in the language of Tuttle, consists "in a large round elevated patch, situated upon a supple base of non-indurated skin. Its color is pearl-white, its surface is smooth and moist, and there is seldom more than one of them about the same anus. They are attached to the skin by a broad, flat base, wider than their summit, and though the skin is supple, there is some redness and inflammation about the attachment."

Ulceration about the anus is a manifestation of secondary syphilis. It is often a continuation of the mucous patch. Sometimes they are

secondary to the abrasions and traumatisms to which this portion of the body is ordinarily subjected. The rectum is likewise subject to this lesion. It is oftener attacked, however, in the tertiary stage, and is then preceded by gummatous growths. The ulcers about the anus occur in the majority of cases about the same time as the cutaneous eruptions. In the majority of instances they show no tendency to extensive spreading or destruction of tissue; in others, however, they are very destructive. Invading the rectum, they occur comparatively late, that is between the periods which we are accustomed to refer to as the secondary and the tertiary.

Gummata of the rectum are rather frequent.

Fournier has described a condition which he has called ano-rectal syphiloma. It consists in a specific fibrous infiltration of the rectal wall, the pathological process beginning in the submucous tissues, and is sometimes followed by ulceration of the rectal mucous membrane. It exhibits a remarkable tendency to contraction. As a rule it produces no symptoms sufficiently obtrusive to direct attention to the parts, until the changes have far advanced. Stricture is a common result of this as of the other syphilitic lesions of the rectum.

Hereditary syphilis very commonly shows its ravages in this portion of the body. Early, it produces ulcerations and erosions, and later various syphilides.

### SYPHILIS OF THE LIVER.

In the liver we find an organ very frequently suffering from syphilis, both hereditary and acquired, but especially from the former. The frequency with which the liver is involved—in nine-tenths of all cases of hereditary syphilis, according to some observers—is not surprising, when we realize the closeness of the relation between the portal circulation, and the circulation of the mother. In acquired syphilis the liver may be affected as follows: (1) Gummata; (2) interstitial hepatitis; (3) perihepatitis. The formation of gummata is the most frequently observed of these. The number of growths in a given case varies greatly, as many as fifty having been found in a single liver. They are usually found in the vicinity of the suspensory ligament. When on the surface of the organ, they may project; when within the gland, they may produce retraction of the surface. The capsule of the liver is nearly always greatly thickened. Sometimes they are associated with a formation of fibrous tissue, which may be limited to their immediate vicinity, or may be diffused through the entire organ. Firm adhesions between the liver, diaphragm and other organs may take place. In interstitial hepatitis, there is a general fibrous formation without the presence of gummata. Then the condition resembles ordinary hepatic cirrhosis, but differs from that condition in the greater size of the connective tissue bands. These



run in all directions, dividing the organ irregularly, and producing numerous surface depressions. The liver in this variety of inflammation has a yellow appearance, and is enlarged and hard. Trousseau has compared the diseased parts to sole-leather. Amyloid changes are sometimes associated with the gummata and the diffuse changes in the liver. Occasionally the fibrous changes take place in the connective tissue along the portal canals and in the capsule of the organ.

In hereditary syphilis, the connective tissue is mainly affected. The organ is greatly hypertrophied. The condition may be either general or partial. In the former case, the liver is flint-colored and hard and inelastic. It may even creak like cartilage on section. In the partial variety, the same changes are manifested, but in less degree. There is narrowing of the calibre of the bloodvessels of the diseased parts. As a result of the excessive connective tissue formation, the liver cells themselves are atrophied. Sometimes the walls of the portal vein are inflamed and thickened.

As in the case of other syphilitic visceral diseases, the symptoms cannot be regarded as characteristic. Physical examination reveals the liver to be greatly enlarged in nearly all cases. Sometimes palpation shows its surface to be nodular, as in carcinoma. In the congenital variety, the symptoms are largely dependent upon the obstruction to the circulation. Umbilical hæmorrhage is an occasional complication, and moderate ascites is sometimes present. Icterus is not common. It is probable, however, that fatal cases with this symptom in infants are dependent upon hepatic syphilis. Sometimes the pathological condition runs its course without a symptom, and is discovered only on autopsy, having even disappeared under treatment.

Pain is often observed in the case of acquired syphilis. It is of a dull, heavy character and is aggravated by any attempt at motion. Jaundice is sometimes observed. Notwithstanding the severity and extent of the interstitial changes, the symptoms are never as prominent as they would lead us to expect. The patient is very often anæmic. Albuminuria and tube-casts may be present. Associated lesions of the spleen may sometimes be noted.

### SYPHILIS OF THE SPLEEN.

This organ may be affected by increased connective tissue formation, or gummata. Aside from the physical signs of enlarged spleen, it may give no evidence of its existence. In other cases we find the cachectic condition associated with ordinary splenic changes.

### SYPHILIS OF THE PANCREAS

gives rise to no symptoms or signs by which its existence may be recognized. Pancreatic disease is rather frequent in hereditary syphilis, and almost unknown in the acquired form of the disease.

## SYPHILIS OF THE KIDNEYS.

During the secondary stages of syphilis, the kidneys are liable to an inflammation which may well be considered as analogous to the nephritis consequent upon scarlatina. The frequency of this condition has been much debated, many considering it one of the rarest manifestations of syphilis. Petersen, who is probably better able to speak on this subject than any other authority, estimates its frequency at 3.8 per cent. It usually occurs during the second or third month after the appearance of the initial sore, though it may set in earlier or later than this date. Its advent is heralded by a suddenly appearing œdema, usually limited to the face and eyelids, though sometimes extending to other portions of the body. Subjective symptoms are remarkable for their absence. The patient may possibly have some general sense of ill-feeling, which may well be attributed to the constitutional effects of the syphilis rather than to localization of disease in any particular organ. The urine presents the ordinary characteristics of acute nephritis. It is scanty in amount, turbid or high-colored, highly albuminous, and not infrequently contains blood. Microscopically, renal epithelia, blood-cells, and granular, epithelial and blood-casts are found. As improvement is established, the granular casts are replaced by others of a hyaline character. Finally all traces of the disease disappear from the urine. While mercury is in this as in other syphilitic diseases of the secondary period, the great remedy, it must be remembered that in just these cases the patients are very liable to show its pathogenetic effects in the mouth.

The tertiary period of syphilis also shows disease of the kidneys, disease of the same type as affects other organs, namely, gummata, chronic interstitial inflammation, and amyloid degeneration. The amyloid degeneration is often believed to be the primary condition, and the interstitial nephritis a secondary pathological development from the same. Kidneys will be found presenting both of these conditions, the amyloid degeneration predominating in some, the interstitial inflammation in others. Gummata of the kidney are very rare. Interstitial nephritis is sometimes associated with these growths, the inflammation being sometimes diffused, though oftener limited to those portions of the organ adjacent to the tumor. Acute nephritis sometimes attacks kidneys already chronically diseased by syphilis. Such cases are of very serious import, the majority dying.

The prognosis of renal syphilis varies with the pathology of the case. Interstitial nephritis offers little or no hope. Much can, of course, be done in the way of palliation and staying the onward march of the disease. Amyloid degeneration may recover under suitable treatment after a long period. Gummata yield readily to anti-syphilitic treatment as a rule, in a most satisfactory manner.

## DIAGNOSIS OF SYPHILIS.

Ordinarily, the recognition of syphilis is a comparatively easy matter. Attention to the details presented in the preceding pages, taken in conjunction with a history of syphilitic infection, is ordinarily all-sufficient. But there are certain questions involved in the clinical study of syphilitic individuals that sometimes make the recognition of syphilis, as the foundation of the patient's ailment, not an easy task. In the first place, there exists the absence of a history of primary infection. It is perfectly possible, as already stated, for the initial lesion to be acquired innocently, and occur on some other part than the genital organs. If it happens to be a dry, scaling papule, it may readily escape coming under the eye of the physician, especially if it be so situated as to cause the patient no inconvenience. In careless individuals it may even escape the observation of its possessor. In a brief time its presence, though recognized as an ordinary cutaneous lesion, may be entirely forgotten. In women the initial lesion, though acquired during intercourse, is very frequently so situated as to be entirely beyond observation, in which case the diagnosis must rest entirely upon the glandular involvement and the secondary and tertiary symptoms. In women, too, another diagnostic obstacle presents itself, and that is the difficulty of obtaining the history of the primary lesions, even when the existence of such is known to them. Whether acquired from looseness in morals, or from an unfortunate choice of a husband, women acknowledge a syphilitic history with the greatest reluctance. In the majority of cases it is wiser with them to make indirect inquiries, *i. e.*, for such prominent secondary symptoms as those of the skin, throat, mouth and hair.

The history of the initial lesion oftentimes lacks in positiveness. When we are told of a chancre followed by enlarged glands and the characteristic eruptions and sore mouth, the syphilitic history is of course beyond question, but where a sore of unknown character has existed, and the secondary symptoms have been slight, the physician is necessarily at a loss for a conclusion. The possibility of mixed infection must always be entertained in the face of a sore possessing the clinical features of a chancroid. The denial of chancre and the admission of gonorrhœa must be entertained as a possible admission of syphilis, providing the present symptoms are indicative of such a state. The supposed gonorrhœa may not have been such, but instead an intra-urethral chancre. Then, too, some patients not willing to acknowledge the whole truth, tell half the truth, trusting that the physician will disbelieve the entire statement. Thus have I had patients admit gonorrhœa without hesitation, and then say they had not had a chancre so far as they knew. The mental state that leads to such deception is hard to fathom. But it exists; and we must face it.



Syphilitic infection admitted, discrimination must be exercised, lest all ills befalling the subject be attributed to the constitutional disorder. While, on the one hand, it must be recognized that syphilis may attack any organ or tissue of the body, it must also be borne in mind that this disease protects from no other constitutional or local disorder, all of which may run their course in the presence of this dyscrasia. The only safeguard against error here lies in a thorough knowledge of general pathology and skill in the application of all the principles of clinical diagnosis. The knowledge that certain symptoms are often of syphilitic origin possesses great value, a value still further enhanced when it is learned that they manifest themselves at fixed periods after infection, periods at which clinical study has taught us to expect them.

One must be careful lest syphilis acquired during childhood escape diagnosis. When one considers the exceeding commonness of this disease, and the many avenues by which infection may be carried, the wonder is that syphilis is not acquired more frequently by children than actually occurs. The danger from syphilitic nurses and their friends is doubtless greater than is generally admitted.

**Prognosis.**—As a rule, the prognosis of syphilis in all of its stages may be regarded as favorable. It is very exceptional, indeed, for the primary and secondary symptoms to do anything else than recover. As to the tertiary manifestations, they must always be regarded as serious diseases, the degree of attendant danger depending mainly on the patient's general health standard and the thoroughness with which anti-syphilitic treatment is carried out. In no class of cases can serious lesions be more thoroughly recovered from than in those of syphilitic character.

While, then, one can give a favorable prognosis as to the ultimate result, the greatest care must be exercised in formulating an opinion as to the duration of present symptoms, their future severity, and the possibility of other syphilitic conditions. No matter how long the period of freedom from symptoms, it is entirely possible for new complications to arise, and this regardless of the mildness or severity of the early stages of the disease. A mild primary or secondary syphilis may be followed by either mild or severe tertiary lesions. It is often taught that mild secondaries predispose to severe tertiary phenomena. The general experience of neurologists favors this view, which has received firm denial by workers in other special fields. If there is any truth in this proposition it probably is found in the fact that the mildness of the early stages leads to carelessness in therapeutics and personal hygiene, with the not unnatural sequel—a bad result.

Individual factors enter so largely into the course of this disease that it is impossible to prophesy without an intimate acquaintance with the patient's personal and family history and constitutional tendencies.

The general course of the majority of cases of syphilis is a mild one, by far the large majority of cases never exhibiting tertiary symptoms. Many clinicians, indeed, have written able articles, advocating the view that, at present at least, syphilis should be looked upon as a benign disease.

Some authorities, prominent among whom stand Gowers and Horsley, place syphilis among the incurable diseases. Theoretically, they may be right. Clinical experience teaches, however, that proper therapeutic and hygienic measures produce amelioration of symptoms tantamount to a cure, and that some few cases so far recover as to make reinfection possible.

For prognostic purposes, syphilitic lesions may be divided into essential and secondary lesions. In the first class are to be placed those pathological conditions arising directly from the syphilis itself. In the latter class are to be included those changes consequent upon the first mentioned. Illustrations of this may be best presented in the case of syphilitic nervous diseases. A syphilitic disease of the cerebral blood-vessels leads to thinning or weakness of their walls, and their rupture. A hæmorrhage is consequent upon this, and this hæmorrhage is for practical purposes, to be regarded the same as a hæmorrhage proceeding from any other cause, subject to the same principles of prognosis and treatment. The essential syphilitic disease here is that of the arteries, and this is susceptible of improvement or cure under proper treatment. To illustrate once more, let us take a cerebral gumma, or a syphilitic meningitis. The mechanical effects of either lesion may lead to local softening or necrosis of brain-tissue. The meningitis or gumma, as the case may be, is curable. The secondary lesion, destruction of brain substance, is not.

The question of syphilis in its relation to marriage is best presented by the conclusions of Van Lennep, as follows: "If a man is free from specific symptoms, and has shown none for eighteen months or two years; if the disease is at least three or four years old, and if not cured, well into the tertiary stage, while the form we know is not obstinate, nor does it show a tendency to endanger the individual; if the treatment has been successful and has been continued after the disappearance of all symptoms—then we can, without hesitation, promise our patient personal safety and children free from this taint. With a woman, the case is different. We should insist on a longer delay of at least a year, and I, for one, should feel considerable hesitancy in consenting to her marrying at any time, if not through anxiety for her possible offspring, certainly for her own future."

**Treatment.**—While the remedial treatment of syphilis resolves itself, in the majority of cases, into the administration of two remedies, namely mercury and iodide of potassium, the successful management of

the disease includes much more than this. When and how to give, the laying down of proper rules of living, and the management of intercurrent ailments, call for the exercise of most excellent judgment. It has already been stated several times that the severity of syphilis is largely dependent upon the general health of the patient. Hygienic precautions must, therefore, be of inestimable value in reducing the lesions to a minimum. They include precautions in every direction. They must be varied from time to time, as this or that organ is threatened with the brunt of the disease. Early in the course of syphilis the mouth is the point of attack. Local lesions here must be avoided. During the second stage of incubation, therefore, the teeth must be put in thorough order. The greatest care in cleanliness must be enjoined. Smoking must be limited as far as possible, for the fumes of tobacco are by their very nature and their heat, irritating to the buccal mucous membrane, and predispose to local outbreaks of syphilis.

The patient must be fully impressed with the danger of his communicating the disease to others, not only by intercourse, but by ordinary social communication.

In the first stage of the disease, that of the initial sore, the treatment should be largely expectant. It is true that radical measures looking to the abortion of the syphilis have been advocated, but they are for the most part inadvisable. Excision of the chancre has been advocated by some few syphilographers. Very little can be expected from this measure, for the disease must be regarded as constitutional even before it is manifest. The claim has been made that even though excision fails to abort, it lessens the severity of the future lesions, a claim that has not been fully borne out by the experience of the best authorities. Cauterizations are likewise useless.

Whether or not to give mercury in the initial stage, is an important question. Here we find great differences in professional opinion. Mercury undoubtedly at this time does tend to prevent or lighten the secondary symptoms. If we succeed in attaining the former of these, what assurance have we that the sore was an infecting chancre? It is well for this reason to avoid such treatment, unless the diagnosis of syphilis is almost mathematically certain. There are circumstances under which it is highly important to avoid secondary symptoms, and under which an abortive treatment of this character may be necessary. These are two: When for family reasons it is desirable to keep the disease a secret and prevent the infection of others; and, secondly, when syphilis occurs in a pregnant woman, and the welfare of the fœtus is at stake.

When, however, general glandular involvement marks the advent of constitutional disturbance, then medication directed to syphilis *per se* must be instituted. This, as already stated, consists in the administra-



tion of mercury in one of its forms. As to which form, individual opinions differ greatly. With the majority of men the preparation is a matter of small moment. I myself prefer one of the iodides, the bin- or proto-iodide. Of these one tablet of the 1x of the former, or two tablets of the 1x of the latter, may be administered three times daily. Their use should be persisted in until symptoms have abated or the constitutional effects of the medicine are manifested. Under no circumstances, however, is the drug to be persisted in to the detriment of the general health. As a rule, it is well to continue the administration of the medicine within reasonable limits until the patient has gone for two years without symptoms.

Ordinarily the administration of mercury by the mouth is all-sufficient. Other methods may exceptionally be required. The hypodermic administration of the remedy seems to be strongly advocated by some syphilographers; but there are few facts to prove that it possesses any distinct advantages beyond the occasional possibility of quicker action.

Mercurial inunctions are sometimes invaluable. I can well recall a case of meningitis, occurring during the secondary stage of syphilis, in which the patient was unconscious, and in which, I have no doubt, mercury thus administered saved a life. The preparation best adapted to this form of administration is the ordinary mercurial ointment—the officinal unguentum hydrargyri. Ordinarily this is too thick for ready use, and it may be diluted with cosmoline. A portion of the mixture, equivalent to one drachm of the undiluted ointment, should be well rubbed in the patient's skin each evening. From twenty minutes to half an hour should be occupied in this process. Different parts of the body may be selected for application each evening; thus, in turn, may be treated the axillæ, the groins, the insides of the thighs, the back, the buttocks and the front and sides of the chest. The inunction treatment is dirty, and is objected to by fastidious patients. Only under circumstances such as that described in the first part of this paragraph is it likely to prove of practical value.

Many physicians advise the conjoint use of hot baths and mercury, and recommend their patients to take a course of treatment at the Hot Springs of Arkansas. Such advice is, to say the least, of questionable value. The time-honored custom among the wealthier classes of resorting to this place for treatment has given the physicians there considerable experience in the management of syphilis, their experience being the main advantage of the resort and not the inherent value of the baths.

After the second year of the disease, iodide of potassium is generally the best remedy. It becomes more certainly so the later the syphilitic manifestations for which it is given. For the nervous lesions, in particular, it is the remedy *par excellence*. But it must be administered in

suitable doses. The suitable dose is the dose that will do the most good. In many of the tertiary lesions, ten grains three times daily will prove sufficient. In the case of nervous and visceral syphilis much more than this quantity is required. Ofttimes it must be administered in doses limited only by the patient's tolerance. It must be remembered, moreover, that the tolerance of syphilitic subjects for this drug is often incredible. The quantities required in nervous syphilis, in particular, are sometimes truly remarkable, as much as from one-half to one ounce of the drug daily being required before a satisfactory result is appreciable. The best plan is to begin with doses of five grains three times daily, about half an hour after meals. On the second day this is increased to ten grains three times daily. On each day, or alternate day, the daily quantity may be increased by thirty grains until at about the end of the first week the patient is taking one hundred and fifty grains three times daily. Then further increase in dosage may be deferred until the action of the remedy has been determined. It is rare, indeed, that a smaller dose than this is curative in the case of nervous or visceral syphilis. It is very rare, moreover, to find these large doses to disagree. Sometimes they do excite an intolerance, but such a result is usually due to faulty methods of administration. Sometimes the dose has been increased too rapidly; sometimes the intolerance is largely imaginary on the part of the patient; sometimes it is due to a bad stomach. The ill effect on the stomach can usually be obviated by administration after meals, the free dilution of the medicine, and the rendering of the solution alkaline by the addition of a few grains of bicarbonate of soda to each dose.

Iodide of potassium is best dispensed in the form of saturated solution, of which each minim represents nearly one grain of the crude drug.

A good result having been obtained, as is usually the case, the use of the drug should be persisted in for a long time after the disappearance of symptoms. The dose, however, should be greatly decreased, only to be added to on the slightest warning of a return of symptoms. It has been said that an exhibition of tolerance on the part of the patient is suggestive of syphilis. While as a matter of fact syphilitic individuals do manifest an ability to take exceedingly large doses of iodide of potassium, this tolerance is not confined to them by any manner of means.

Other remedies, notably *aurum*, *thuja*, *sulphur*, *silicea*, *asafoetida*, *nitric acid*, *arsenicum*, *hepar*, *iodine*, *fluoric acid*, and *mezereum*, are of value for special indications, which are found in all works on materia medica.

If the above plan of treatment strikes the reader as unnecessarily crude, it is only necessary to remind him of two facts: First, that the results from it are good, and secondly, that mercury and iodide of potassium are both homœopathic to syphilis, and the dose as a matter of therapeutic principle becomes a secondary consideration.

If also the reader feels that too much attention has been devoted to the consideration of syphilis, let me say that this is a very common disease, in some communities in our own country not less than 5 per cent. of the population being the victims thereof. If then the commonness of the disease and the magnificent results to be obtained by proper treatment are borne in mind, it will be felt that the space occupied has not been taken in vain. One recent author announces lack of experience in the treatment of syphilis, as his class of patients were of too high caste to have such a disease. But then high birth and station are not prophylactic against bad morals, and squalor and idleness are no less efficient; the wives and children of the transgressors have the effects of the sins their ancestors visited upon them—poor, innocent sufferers. No matter what the present state of a man's morals may be, however influential he is now in society, state, or church, he may once have been young and foolish, and exposed himself to the dangers of syphilis, and, as Gowers has so tritely put it, "no man who has once exposed himself by promiscuous venery can assure himself that he has not syphilis."

### HEREDITARY SYPHILIS.

**Synonyms.**—Congenital syphilis; syphilis hereditaria; infantile syphilis. The latter term should be regarded as inexact, for while the majority of cases of syphilis in infants are inherited, sight must not be lost of the fact that infants and children may become infected and acquire the disease in a number of ways.

In the study of this subject, it must be borne in mind that we have to deal with the same conditions as in the acquired form, the clinical distinctions between the two arising (1) from the absence of a primary stage (initial lesion); (2) the age of the patient; and (3) the fatal character of the lesion.

**Etiology.**—Hereditary syphilis, as the name implies, is that form of syphilis in which infection takes place prior to birth by reason of the existence of the disease in either one or both parents. This statement at once suggests a consideration of a number of points, all of which have a practical bearing. The first suggested is the age of a parental syphilis before all danger of transmission to the offspring has passed. In a general way it may be stated that during the secondary stage of the disease, and for two years after there have been active manifestations, the chances are strongly in favor of transmission. It is as yet regarded as doubtful whether there is a possibility of children inheriting the disease from parents manifesting tertiary symptoms. Cases favoring the affirmative of the dangerous character of the latter to the infant have been recorded, but for the most part lack authenticity. Exceptional instances have been reported in which conception has taken place while one or both parents exhibited active secondary syphilis, and yet the infant escaped.



Such cases, however, are very rare indeed. On the other hand, inheritance has taken place when the disease has remained latent for as long as fifteen years.

The syphilitic infection of a pregnant woman will be transmitted to her child, providing the syphilis has been acquired at a sufficiently early period to permit of the development in her of symptoms indicative of constitutional infection. The disease thus becomes dangerous to the foetus if acquired at any period prior to the eighth month of pregnancy. The possibility of infection of the infant by the mother's discharges during birth, is here raised. This rarely occurs, probably because the child is well protected by the *vernix caseosa*.

The efficiency of anti-syphilitic treatment of the parents undoubtedly exerts a great influence in the prevention of syphilitic inheritance. Case after case has been reported, in which absolute freedom of the infant from the disease has been secured by this means alone.

It is generally conceded by syphilographers that syphilis may be inherited from the father alone, the mother remaining healthy. This fact has been strenuously denied by Sturges and some others, who have advanced very ingenious arguments proving the falsity of such a position. He claims that the mere fact that the mother gives no evidence of the disease after repeated thorough physical examinations, is without value. Syphilitic children, he says, continue to be born, although the father be rigorously treated; while treatment of the mother is also effectual as a preventive of transmission to the child. He firmly believes that in every case where the mother remains healthy, the child does not become infected. The arguments in favor of this position are after all theoretical; for, practically, we find the truth to be with the majority of those who have given especial attention to the subject.

A syphilitic infant thus born cannot infect its mother, although it is capable of readily infecting any other woman who may act as its wet-nurse. This statement is known as Colles's law. It is believed that through some means the mother becomes immune, and thus escapes the disease. Diday and Bouchard offer a very reasonable explanation that is doubtless correct. All infectious diseases, say they, may be mitigated to the point of absolute protection by the methodical repeated inoculation of their essential cause, or of its products. Bouchard says that while the foetus retains the supposed pathogenic agent itself, the products dissolved in the blood find their way to the mother, and set up nutritive changes, resulting in what he calls a bactericidal condition. It renders difficult or impossible the development of the infectious agent when introduced later by inoculation, as from the lips of her own child.

In the same way children born of syphilitic parents, but not infected by the disease, may acquire an immunity from acquired syphilis. This phenomenon is known as Profeta's immunity.

The ability of syphilis in the mother alone to produce the disease in the offspring, has not been questioned by any syphilographer. It is generally conceded that the disease transmitted from this source is apt to be of a more severe grade than when inherited from the father, and that it is still more severe when both parents are affected. Hutchinson denies the truth of these latter statements, claiming that the source of the inheritance does not exert the slightest influence on its intensity. He admits, however, that syphilis in the mother is somewhat more likely to make itself felt in the child than is the disease in the father.

One or both parents being syphilitic, the child has the best chance of escaping as the interval between primary infection and conception increases. A systematic course of anti-syphilitic treatment during pregnancy will do much towards lessening the danger to the infant.

Women pregnant with a syphilitic fœtus are exceedingly liable to abort, especially so if they themselves happen to be the subjects of secondary manifestations. The abortion is especially liable to occur at about the third or fourth month, the fœtus being born macerated. Miscarriage may take place at any period of utero-gestation, however.

The severity of the type of the affection in the parents has little or nothing to do with the severity of that in the children. A mild parental syphilis may beget a severe, hereditary form.

It is generally conceded that the children born of a syphilitic parentage exhibit less and less evidence of the disease with each succeeding pregnancy. Thus, the first pregnancy may result in miscarriage with a macerated fœtus at the third month; the second, in miscarriage at the sixth month; the third, in a still birth; the fourth, in a living child which shortly gives distinct evidence of syphilis; a fifth, in a child which exhibits the lesions at a still later period; while subsequent children remain entirely free from evidences of the disease. Hutchinson denies this most positively, and claims that healthy children are so, because they happen to be conceived and carried during quiescent stages of the syphilis, and that subsequent ones may be as severely affected as the first.

The possibility of the transmission of syphilis to a third generation has been discussed and decided in the negative. Some few cases claimed to have been thus transmitted, have been reported; but careful analysis shows important links missing in the chain of evidence.

**Clinical Course.**—The period at which evidences of hereditary syphilis assert themselves is tolerably well defined. In practically all cases this is before the sixth month, and, in nearly all, before the third. Diday's statistics gives a good idea of the periods of onset. Of 249 cases, symptoms appeared in 118 before the first month, 117 between the first and third months, and only 14 after the third month. Late hereditary syphilis—syphilis hereditaria tarda—*i. e.*, hereditary syphilis in which the initial manifestations appear from the tenth to the twentieth

year of life, has been described. The best syphilographers are of the opinion, however, that in these cases the early symptoms of the disease have been ignored or forgotten.

The frequency with which a child may be born with active syphilitic manifestations is a debatable point. The careless phraseology of many writers would lead one to believe that this is of frequent occurrence. Hutchinson, on the other hand, says he has never seen such a case, and other leading authorities make it of decidedly exceptional occurrence. When syphilis begins in utero, and early miscarriage does not ensue, the child is pretty certain to be still-born.

The initial symptoms of hereditary syphilis are those of the skin and mucous membranes. The former usually consists of an erythematopapular or roseolar eruption, appearing commonly about the buttocks. Sometimes there are rhagades about the mouth, especially about the upper lip. The earliest evidence of mucous membrane implication is coryza. The discharge is watery and excoriating. The mucous membranes of the mouth and throat sometimes exhibit mucous patches, and the laryngeal mucous membrane becomes congested, a condition that is evidenced by the peculiar harsh and irritating quality of the child's cries.

The general appearance of the skin, even when free from eruption, is bad. It is dry and inelastic. The cutaneous appendages, the hair and nails, exhibit the same changes as in acquired syphilis. The child is nearly always in a marasmic condition, and presents an appearance which has not inaptly been described as similar to that of an old man.

General symptoms are nearly always present. The appetite is poor. Digestion is decidedly disturbed. Vomiting and diarrhoea are frequent.

The lymphatic glands in different portions of the body may be enlarged. This symptom is less characteristic of inherited than of acquired syphilis. Parrot looks upon the glandular enlargement as secondary to the eruptions. Post, on the other, has seen numerous cases free from skin diseases in which this symptom was present.

Post and Eustace Smith direct attention to a symptom very generally ignored by physicians, and that is wakefulness. It is one of the earliest evidences of the disease. Night after night the child is uneasy and wakeful, and may cry unceasingly. During the day he seems to be tolerably comfortable. This phenomenon finds its explanation in probable nightly bone pains, such as commonly occur in acquired syphilis.

Very often the eruption of the teeth is greatly delayed in syphilitic infants.

Iritis is an infrequent symptom. Still it has been observed twenty-three times by Jonathan Hutchinson. In his cases the period of its appearance ranged from the sixth week to the sixteenth month.

Later in life the subject of hereditary syphilis very often gives



marked objective evidence of the disease. Prominent among these in some cases is the condition described by Fournier as "infantilism." The subject gives every evidence of immature physical and sexual development. The disposition is that of one of fewer years, and the mental powers are those of a mere child; in the youth, the testicles remain small and the beard fails to grow. In the girl, the mammæ fail to develop and menstruation is delayed long beyond the usual age of puberty. This failure in development is readily foreseen in early childhood by the slowness with which the little one learns to walk, talk, and progress as normal infants should.

The nasal deformity of hereditary syphilis is very characteristic. It results from disease of the bones and cartilages making up its framework. The result is a falling in of the bridge. This condition must not be confounded with congenital malformations. In syphilitic cases it is always the result of previous disease of the bones and cartilages, which can be discovered by proper examination.

The permanent teeth very often exhibit changes which are by many regarded as pathognomonic of congenital syphilis. Syphilitic teeth were first described by Mr. Jonathan Hutchinson, and are often referred to as "Hutchinson teeth." They may best be described in his language: "The upper central incisors are dwarfed, too short and too narrow, and if they display a single central cleft in their free edge, then the diagnosis of syphilis is almost certain. If the cleft be present and the dwarfing absent, or if the peculiar form of dwarfing be present without any conspicuous cleft, the diagnosis may still be made with much confidence. Usually the conditions are symmetrical, but now and then they are notably one-sided. It is remarkable that the lateral incisors rarely show any peculiarity, but they are also sometimes dwarfed. The peculiarities in other teeth (in the lower incisors, the canines and the molars) are, so far as I know, of a kind which it is impossible to distinguish from those due to stomatitis. It would be well if the term pegged and peg-shaped, or, as some absurdly write, peg-top teeth, were disused." "The lower incisors not infrequently show a dwarfed and foliated condition . . . due to syphilis and not to mercury; but these conditions are not to be trusted in the absence of peculiarities in the upper central incisors."

A very characteristic late eye symptom is interstitial keratitis. This usually appears between the sixth and twelfth year; but it has been observed during infancy, and as late as the age of twenty-five.

The nose, throat and pharynx are favorite seats for the late manifestations of hereditary syphilis, these consisting, in the main, of deep ulcerations.

The bones and joints are very frequently involved. Especially are the tibia, radius, ulna, femur, humerus, clavicle and sternum liable to disease from this cause. The pathological lesions are periostitis, osteomyelitis and osteo-chondritis.

The remaining phenomena of hereditary syphilis have been sufficiently described in the portion of the preceding section devoted to visceral syphilis.

**Diagnosis.**—The presence of a cachectic constitution in the child, and an acknowledgment of acquired syphilis in one or both of the parents, is all-sufficient for diagnosis in most cases. Often, however, the latter is not forthcoming, and one must depend entirely upon the clinical phenomena presented. Here a difficulty is encountered, because there is no one symptom which can be considered as pointing indubitably to syphilis. One must be guided by a conjunction of symptoms. Certain symptoms are, of course, strongly suggestive, such as the eruption about the buttocks and anus, pemphigus, onychia, the “snuffles,” the ulceration of the mouth, and the peculiar harsh cry.

Later in life, Hutchinson’s trio of symptoms, the teeth, the configuration of the nose, and the interstitial keratitis, should be regarded as positive proof. Some physicians have claimed that all three of these symptoms can be present in non-syphilitic subjects. In view of the many confirmations of Hutchinson’s observations, such assertions must be regarded with suspicion.

The great danger in the diagnosis of hereditary syphilis lies on the one hand in ignoring mild though unequivocal symptoms, and on the other, of attaching altogether too much importance to single symptoms, *e. g.*, the ordinary aphthous sore mouth of infants. It must be remembered that a symptomatic group is required to make the complete picture of this disease.

**Prognosis.**—Inherited syphilis must be regarded as a very serious disease. A very large proportion of the cases end in early abortion. Very interesting statistics are at our disposal. Julien observing 206 cases, finds that 36 ended in abortion, 8 in still-birth, and 69 died at an early age. There were 93 living children, of whom 50 were syphilitic. The deaths in early life were as follows: Meningitis, 21; convulsions, 8; throat diseases, 12; diarrhœa, 5; other diseases, 23. Kassowitz states that one third of syphilitic children dies before birth, and another third before the age of six months. Fournier reports 103 cases, with 84 abortions, premature births, or early deaths. Hochsinger was enabled to keep 63 out of 265 cases under observation for over four years. The following were his conclusions: (1) Ten showed syphilitic ulcerations within the time specified. The oldest of these was twelve years of age. (2) Another group comprised eighteen cases, who remained in health in every respect. (3) The remaining twenty-five showed no signs of syphilis, but some of them have the visible marks of hereditary affections, and others were affected with general or undetermined morbid conditions. None of the cases showed any direct connection between syphilis and scrofula. As to the forms of relapses, during the first few years of

life, the cases showed, almost exclusively, condylomatous eruptions of the skin and mucous membranes; during the fourth and fifth years, gummatous ulcerations or bone diseases as well as eruptions; beyond this time, only gummatous changes.

Certain general principles guide us in framing a prognosis. This is the more favorable as proper treatment is instituted early, and as the interval between infection of the parent and conception is longer. Syphilis of the mother has a more deleterious influence than that of the father; and syphilis of both parents is still more serious. Post-conceptional syphilis is the more serious, as it is acquired earlier in the pregnancy.

**Treatment.**—In the treatment of syphilitic infants every hygienic rule must be most carefully observed. Nothing is more important than the diet. If at all possible, the child should be nursed by its mother. Objection has been made to this, because of the deterioration of her milk by her syphilis. Even with this drawback, the mother's milk is certainly better than any artificial preparation. The substitution of a wet-nurse is of questionable propriety. If she be a healthy woman, she will almost certainly acquire syphilis from the nursling. If she be employed, she should be informed of the dangers she incurs. There can be no objection, of course, in employing a wet-nurse who has already had syphilis, and who does not give at the time any active manifestations of the disease.

The medicinal treatment of hereditary syphilis resides mainly in the intelligent administration of mercury. The preparations available are *mercurius dulcis*, *mercurius corrosivus*, *mercurius biniodide*, and the officinal mercurial ointment. As soon as symptoms appear, one of these remedies should be given. *Mercurius dulcis* 2x, one tablet every two or three hours, may be tried. If it produces too lax a condition of the bowels, its use must be abandoned. *Mercurius corrosivus* 2x may be given in doses of one tablet, three or four times daily.

The inunction treatment of syphilitic infants is in many cases preferable, and is very easy of application. From one to two drachms of blue ointment are spread upon a flannel binder, which is then wrapped about the child's body. This is permitted to remain for two or three days, when it is removed, the skin carefully washed and the ointment reapplied. The administration of mercury should be stopped very shortly after the disappearance of symptoms.

*Aurum*, *nitric acid* and numerous other remedies may be indicated from time to time, as mentioned in the section on the treatment of acquired syphilis.

For the late manifestations of hereditary syphilis, iodide of potassium is the main remedy. It is to be administered in the same way as in acquired syphilis.



## LEPROSY.

**Definition.**—An infectious disease of chronic type, characterized by a nodular infiltration of the skin and mucous membranes, and frequently by inflammatory lesions of the nervous system. A specific micro-organism, the bacillus lepræ, is constantly present.

**History and Causation.**—Leprosy was known to the ancients. Hippocrates called it lepra. It was also known as elephantiasis. It is spoken of in the Bible as Zaraath. With the fall of Rome, knowledge of the disease disappeared. After the eighth century the disease reappeared upon the European continent, the crusaders returning from the East being chiefly instrumental in its spread. The twelfth and thirteenth centuries witnessed its greatest prevalence. In modern days we find its geographical distribution to be quite extensive. In India, Norway and Sweden, New Brunswick, Mexico and the Sandwich Islands, the disease is common. A quarter of a million of lepers are said to exist in the first-mentioned country. In America cases are occasionally found among foreigners, especially along the coast regions. On our Pacific Coast it prevails almost exclusively among the Chinese. On the eastern shore of North America there is a noted colony of lepers at Tracadie, New Brunswick. A few cases have been found in the Gulf States, and at Key West. The disease is endemic in the West Indies. Excellent contributions to the literature of the subject have been made by Rake, of Trinidad.

The bacillus of leprosy was discovered by Armauer Hansen, of Bergen. It presents morphological characters similar to those of the bacillus of tuberculosis. "The bacilli are fine rods about 0.004 to 0.006 millimeters long and less than 0.001 millimeters thick. They are pointed at their ends and always occur in masses within the large leprosy cells of the leprous tubercles of the skin and internal organs" (Klein). Neisser, who has increased our knowledge of this subject, has cultivated the bacillus in blood serum and meat extract, and also shown that the "leprosy cells" are migrating cells, stimulated to change by the development of the bacillus within them. Successful inoculation of animals has not yet been performed, although Arning claims to have succeeded in infecting the Hawaiian criminals. The disease is believed to be contagious, but the method of communication is unknown. That it is not readily communicable is shown by the infrequency with which nurses and even children of the afflicted are infected. Its contagiousness has been compared to that of syphilis, yet there is no evidence of a primary point of infection. Some observers teach that sexual intercourse

is the almost invariable means of its communication. Others affirm the opposite. Hereditary transmission seems probable, but this doctrine does not harmonize with modern ideas of its cause. Fagge states that there is not a congenital case on record. A fish diet has been looked upon as a predisposing cause, so good an observer as Jonathan Hutchinson advocating this view. Plausible as it seems, we think that this position cannot be maintained.

It is claimed that leprosy has been communicated apparently by vaccination.

The most frequent age of infection or development appears to be before the twentieth year, although cases occur until the age of forty, when the disease diminishes in frequency.

**Clinical Course.**—Leprosy is very insidious in its onset. Its symptoms vary much according to the localization of the lesions. The most superficial tissues are most frequently involved primarily. They become the seat of infiltrations, which appear as nodular elevations upon the skin (*tubercular leprosy*). In a second form, the infiltrating process primarily attacks individual nerve-trunks, with resulting sensory, motor, and trophic changes in the tributary parts (*anæsthetic leprosy*). The different types of leprosy are often found mixed in actual practice, the anæsthetic form supervening upon the tubercular. Leprous lesions may develop on the mucous membranes, especially those of the pharynx, larynx, trachea, cornea, and conjunctiva. They are also found in the liver, spleen, lymphatic glands, testes, etc.

**TUBERCULAR LEPROSY.** Preceding the development of this form of the disease, a period of a year or more, in which occur symptoms simulating those of intermittent fever, has been observed. Locally, and prior to the development of the nodules, areas of erythema develop. These may be hyperæsthetic. The color may ultimately disappear, a white spot remaining; in other cases, a brownish discoloration replaces the erythematous blush. These spots occasionally become anæsthetic. The nodules develop gradually, becoming hard, and finally appearing as large knobs and variously shaped elevations. The affected region is consequently thickened, disproportioned, inelastic, and may finally ulcerate. The progress of the disease is not uniform at all points, some of the nodules ulcerating, and others disappearing. Changes, similar to those above described as appearing on the skin, may involve the mucous membranes; but the cutaneous usually precede the mucous lesions by a considerable period of time. To the above may be added, in certain cases, the symptoms caused by leprosy lesions in the eyes, nose, throat, larynx, lymph-glands, and internal organs. Loss of hair, especially from the eyebrows, is common.

**ANÆSTHETIC LEPROSY** varies in important particulars from the tubercular variety. The nodular infiltration of nerve trunks may be

felt, if sufficiently superficial. Areas of hyperæsthesia, which, later, become anæsthetic, dry and dull in appearance, and which may be preceded by bullæ, constitute the earlier group of symptoms. To these, there are added, later, marked trophic changes. Pemphigus-like bullæ may form; muscles waste and contract, and some degree of paralysis ensues. Destruction and loss of toes and fingers, and even of entire extremities, result. In this form, the deformity is less.

The duration of leprosy may be many years. Death results from exhaustion, putrid infection, damage to various organs, etc., or from intercurrent disease.

**Morbid Anatomy.**—Virchow, Thoma, Kaposi and others have carefully studied the lesions of leprosy. The tubercles gradually extend to and involve the skin, and consist of granulation cells in a reticulum of connective tissue. Ulcerative changes lead to loss of substance, even of fingers and toes (*lepra mutilans*). The new growths appear upon parts most exposed, *i. e.*, the face, hands, and feet. The bacilli are found in large number in and between the cells of the new tissues. A peripheral neuritis, excited by the bacilli, is the cause of the sensory, motor and trophic changes of anæsthetic leprosy.

**Treatment.**—While a case of spontaneous recovery has been reported by Hutchinson, leprosy is regarded as practically incurable. Removal from an endemic centre, and the securing of the best hygienic surroundings for the patient, assist in retarding the course of the disease. Good food, fresh air, the internal use of certain oils, such as cod-liver oil, chaulmoogra and gurgelon, with careful dressing of the leprosy ulcers, have given the best results. As to the medicines to be recommended, there is not sufficient evidence of their value to even demand an enumeration of them.



## WEIL'S DISEASE.

**Synonyms.**—Acute infectious jaundice; acute febrile jaundice.

This disease was first described by Weil, in 1886. Its nature is as yet obscure, hence the name, "Weil's disease," is probably the best for present use.

**Etiology.**—Weil's disease occurs in isolated instances or in small epidemics, affecting males chiefly, and preferably those between the ages of fifteen and thirty years. It occurs most frequently during the summer months, and in communities in which drainage is defective or the inhabitants are exposed to emanations from putrescent materials. In the epidemic observed by Jaeger, of Stuttgart, seven out of the ten patients had bathed in the river, the water of which was contaminated.

**Pathology and Morbid Anatomy.**—Jaeger's investigations disclosed the presence of a short, slightly curved, rod-like organism, provided with cilia, capable of growing in various culture media, to which it imparts a greenish fluorescence. To this bacillus he proposes to apply, the name "*bacillus proteus fluorescens*."

The pathological changes found in fatal cases are fatty degeneration of the liver and kidneys, interstitial small-celled infiltration of the liver, acute parenchymatous nephritis, hæmorrhages into various structures and splenic enlargement.

**Symptomatology.**—The onset of the disease is sudden, being ushered in by a chill, quickly followed by fever, during which the temperature rises rapidly to 104° or 105° F. It remains at this high point until from the fifth to the eighth day of the disease, when it begins to subside gradually, reaching the normal on about the twelfth day. The pulse, however, does not keep pace with the fever, ranging usually from 100 to 110 per minute. With the appearance of the jaundice it usually decreases in frequency. Jaundice is usually early in making its appearance, and varies greatly in intensity. It is accompanied by enlargement of both liver and spleen and clay-colored stools. The urine contains albumin, and microscopic examination reveals the presence of hyaline and epithelial casts, and red and white corpuscles. In some few cases cerebral symptoms, as delirium and coma, are observed. In about a fourth of the cases there is a recurrence of the symptoms at the end of a week.

**The Prognosis** is as a rule favorable.

**Treatment** must be conducted on symptomatic indications.

## THE PLAGUE.

**Nomenclature.**—The pest; the bubonic plague; inguinal plague; Oriental plague; Levantine plague.

**Definition.**—The plague is a specific contagious fever, endemic and epidemic in Eastern countries, and characterized by the appearance of buboes, carbuncles and petechiæ.

**History.**—Owing to the carelessness with which the term plague was used in ancient times, the history of this disease is involved in some obscurity. Its confusion with other epidemic ailments is generally admitted. It is known that it prevailed in Egypt, Libya and Syria prior to the Christian era. It became widely prevalent in Europe during the sixth century, starting in Egypt about 542 A.D., and spreading rapidly in every direction. Before the close of the century, fully one-half of the inhabitants of the districts visited died, either as the direct result of the disease or of the famine and desolation attendant upon such a fearful epidemic. From that time the disease prevailed in Europe continuously for a thousand years, when it began to die out. During the past two hundred years it has been limited to Asia and Africa, excepting in a few instances, when Turkey and portions of Russia were invaded. Within the past thirty years it has existed mostly in western Arabia, China and northwestern Africa. At the present time it is epidemic in China.

**Etiology.**—The specific cause of the plague is undoubtedly a micro-organism, the nature of which is at present unknown. All other agencies favoring the occurrence of the disease must be looked upon as predisposing only. Prominent among these stand poverty and famine, with the attendant bad hygiene found among the people thus exposed. Accumulation of filth about dwellings undoubtedly favors the spread of the disease. The season of the year seems to have but little influence, as the plague has continued throughout hot and cold weather, although it seems to be less malignant in hot, dry periods, and more active in the midst of dampness. It is especially liable to attack individuals below the age of fifty years.

As is the case with typhus fever, the “striking distance” of the poison is not great. The contagious principle is undoubtedly disseminated through the air. The danger of contagion is directly as to the amount of exposure.

**Symptomatology.**—The stage of incubation ranges in duration from two to seven days; so far as is known, it is never more than eight.

According to the severity of the attack, cases have been divided into the (1) grave or ordinary form; (2) the fulminant; and (3) the abortive.

The ordinary form of the disease sets in with symptoms of a severe type, such as mark the onset of other malignant infectious diseases. They consist of pains in the back and extremities, general prostration, headache, vertigo, nausea, vomiting and diarrhœa. Then comes the fever, which is characterized especially by its high range. In the majority of cases it fluctuates between  $102^{\circ}$  to  $104^{\circ}$  F., but not infrequently reaches  $107^{\circ}$  F. The pulse is generally from 120 to 130. The patient soon becomes delirious, and goes into the so-called typhoid state. The buboes then make their appearance. About this time there is a rapid fall of temperature, even to several degrees below the normal. This crisis is accompanied by a profuse sweat of a strong odor, the pulse becomes weak and the mind clear.

The buboes are especially prominent in the inguinal region and the upper portions of the thighs; sometimes they appear in the axilla and behind the angle of the jaws. They vary greatly in size in different cases; they undergo rapid suppuration and discharge an ichorous pus.

In favorable cases convalescence sets in from the sixth to the tenth day; it is often prolonged by the profuse suppuration.

In the fulminant form the patient is stricken down with all the manifestations of a profound systemic poisoning, as in the cases of cerebro-spinal fever of the foudroyant type, and dies within a few hours.

The abortive cases are characterized by the same symptoms as those observed in the ordinary type of the disease, but which are noteworthy because of their mildness.

**Prognosis.**—No epidemic disease is as fatal as is the plague. During the early days of an epidemic nearly all affected die; later, the prognosis is not so universally unfavorable, although even then over one-half of those attacked die.

**Treatment.**—Nothing can be said respecting treatment, as no epidemics have been observed by educated physicians within the past few decades.



# DISEASES OF THE NERVOUS SYSTEM.

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## GENERAL REMARKS ON DISEASES OF THE NERVOUS SYSTEM.

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The difficulties attendant upon the clinical investigation of diseases of the nervous system are greatly enhanced by the prevalence of false ideas concerning the study of neurology and the examination of patients. The most accurate results are obtainable when the clinician is in possession of a thoroughly practical knowledge of the anatomy, physiology and pathology of the nervous system, and a well-trained, practical mind with which to apply his knowledge. The same remark is equally true in the practice of other branches of medicine, but the difficulties are not so apparent. It is generally regarded as a simple matter to diagnose a pulmonary inflammation, a valvular abnormality of the heart, or an indigestion. But given a cerebral inflammation, a spinal degeneration, or a neural disorder, and puzzling clinical problems are forced on the observer. The pathological changes in the nervous system are no greater in number than those found in other portions of the body, but the symptomatic phenomena differ widely, according as this or that set of fibres or group of cells is the seat of disease. When, as is often the case, different sets of fibres and cells are simultaneously or successively involved, the symptomatic picture may be made very confusing. This confusion is to be avoided by a careful analysis of all the symptoms of the case, and the proper clinical value attached to each by the knowledge of anatomy, physiology and pathology applied by the laws of evidence. Nervous diseases depart greatly from fixed types. This statement applies alike to both organic and functional disorders, especially, however, to the latter class. To attempt, in many cases, to match the case in hand with definite text-book descriptions is futile. Accuracy in results is obtainable only by thoroughness in methods of examination and the "tracking" of symptoms. I cannot illustrate the value of "tracking" symptoms better than by relating an incident in ordinary life. One winter's day, a few years ago, a medical friend called on me. It was snowing. When he entered he said: "You have been home but a

short time, there was a man waiting for you when you arrived ; it did not take you long to get rid of him ; and he has been gone but a few minutes. I did not see you or him. How did I find all this out ?” and then he smiled at my puzzled look of inquiry. Expressing my ignorance as to how my friend obtained such accurate information, he continued : “ There were marks in the snow showing that a man had walked up the step, had rung the door-bell, had stood for a short time, and had been admitted. Following his footsteps were fresher ones starting from the curb. Their owner went directly up the steps, did not pause at the bell-pull, but went straight to the vestibule door. He was therefore in possession of a latch-key. I guessed that he was you. Then still fresher footsteps were seen directed down the steps. These were of the same kind as those preceding you, so I guessed the party had come in to see you, had accomplished his errand and departed.”

Not far from my office practises a quack whose claims for an ability to diagnose cases without asking questions are widely advertised. He gives lectures to the public at stated intervals. On one of these occasions he said, boastingly, that he could tell what was the matter with any one in the room by simply looking at him. A man standing up in the rear of the hall said, “ You cannot tell what is the matter with me.” The quack at once replied, “ You have a rupture.” The correctness of the opinion so startled the patient that he at once offered a fee for knowing the means by which the lecturer obtained this information. “ Easy enough,” said the quack, “ you have on a badly-fitting truss, and while you were standing there I observed you adjusting it from time to time.” The old saying, “ Straws tell which way the wind blows,” is applicable in medicine. Each little peculiarity in the patient’s illness and personality should be correctly observed by us, and the proper interpretation placed upon the same.

So it is with symptoms. They are infallible signs when properly interpreted. But they are very frequently misinterpreted, because they are studied unmethodically and illogically. To furnish the most complete information, they must be stated in their chronological order and they must be described as they appeared. Their association with other symptoms must be most carefully noted. In the taking of records there is altogether too common a practice of noting each individual symptom of a case on a separate line, robbed of all its associations. Such a practice in record-taking cannot be too severely condemned. As to a good anamnesis of a case, it is like a mixed-up heap of bricks and mortar to a nicely finished ornamental brick wall. At this moment I have before me the record of a case concerning which my opinion is sought. Each symptom is very lucidly stated so far as the mere words describing that particular symptom are concerned. But each symptom is disconnected from all others. No note is taken of symptoms that once were, but now

are not. The time of onset of not one is stated. And yet as I look over the record, I can appreciate the labor spent by the physician in taking these notes. But his efforts were misdirected.

How then shall a case be examined? In the first place, the age and occupation of the patient should be noted as a routine measure. Both of these are valuable data oftentimes as enabling one to frame both diagnosis and prognosis. Next comes the history of the case. In starting this latter it is bad practice to ask the patient "What is the matter with you?" In the majority of cases he will answer you in one of two ways: "That is what I came to you to find out," or he will give you his own diagnosis, which is far more likely to be wrong than right. In either case your question has been wasted. "What do you feel that is wrong with you?" "How do you feel badly?" are legitimate questions and do well as first inquiries, provided they are properly followed up. My own practice is to first determine exactly when the patient's health began to show signs of impairment. Receiving definite information on this point, I then ask for a statement of the evidences of bad health then occurring, together with an account of all illness or bad feelings from that time to the present. If the patient keeps himself strictly to facts, and does not talk too much on side issues, it is best to let him tell his story without interruption, the physician in the meantime taking full notes.\* The history completed, the physician should start from the beginning of his record, asking questions to bring forth more clearly the true meaning of the patient's statements. Such expressions as "rheumatism," and "nervousness" have very different meanings with different patients. The lightning pains of ataxia, the pains of neuritis, neuralgia, myalgia, muscular strain, periostitis, cellulitis, etc., may be described by the patient under the former title; and under the latter may be included tremor, restlessness, worried feelings, and any number of indescribable sensations. Even such a positive symptom as pain cannot always be relied upon, for patients often thus designate mere uncomfortable sensations.

In the making of the anamnesis of a case, there are certain data which should never be omitted. I have already stated age and occupation. In addition to these, there are also necessary statements as to the chronicity of the case as a whole and of each of its symptoms, the mode of onset, its apparent causes, the family history, and the history of preceding illnesses, habits as to alcohol, tobacco, venery, eating, drinking, sleeping, working, and recreation.

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\* Here comes in the value of shorthand to the physician. I will be pardoned for bringing this matter in at this place, I trust. Shorthand is very readily acquired. One month spent in the study of the signs and a determination to put the knowledge thus acquired to practical use will be found amply sufficient, for the practical purposes of a physician.



Certain diseases occur almost exclusively in childhood, as chorea, acute poliomyelitis, and Friedreich's ataxia. Others appear in advanced life, as the degenerations of the brain and spinal cord. Some organic affections are especially characteristic of young adults, as syphilitic disease and disseminated sclerosis.

The influence of occupation is shown in the colic, the wrist-drop, the optic atrophy, and the encephalopathy of lead-workers; the writers' cramp of clerks; ailments arising from exposure generally in soldiers; lithæmic conditions in persons of sedentary habit; worry and nervous exhaustion in brokers, physicians, and professional men generally.

Indulgence in alcohol may produce nervous conditions either indirectly or directly. Indirectly, by disordering the stomach, liver, and other organs; or directly, by its specific action on the nervous system. In the latter case it produces tremor, delirium tremens, neuritis, and other conditions, which will receive full elucidation in subsequent pages of this work.

Tobacco produces mostly functional disturbances, exerting its principal baneful effects on the heart, nerves, and throat. Under ordinary circumstances it is capable of doing harm without producing any subjective phenomena. Careful observation shows, however, that the habitue is less able to withstand extra work thrown on his heart after using the weed. Neurasthenic conditions, as a rule, arise only from long-continued and excessive use, especially of cigarettes.

Sexual excesses are productive mainly of functional nervous disturbances. I am not satisfied that I have ever seen a case of organic cerebral or spinal disease due to this cause. Masturbation, undoubtedly, exerts a more deleterious influence than sexual intercourse, probably, however, owing to the attendant circumstances. It is practised at an age when the system can least withstand such debilitating losses as frequent seminal discharges; it is practised more frequently than intercourse; and its unfortunate victim, owing to his weakness and folly, gradually forfeits his self-respect.

Excessive indulgence at the table produces a variety of nervous conditions, especially if the "glutton" is not accustomed to exercise. Too free indulgence in any particular direction also has its bad effects. The extravagant meat-eater is liable to gout, lithæmia, and the nervous symptoms of renal inadequacy. The one who relies almost exclusively on starchy foods, as rice, bread, etc., finds his nervous system gradually undergoing deterioration. Certain articles indulged to excess habitually, as tea and coffee, have specific injurious influences of their own.

A study of the patient's habits as to work and recreation often gives the clue to the difficulty. The human machine may work under high pressure for years without giving evidence of wear; but with the majority of men there comes a time when abused nature rebels against such

treatment. The capacity of different men for work varies greatly. Some thrive under burdens that will kill others. Overwork exerts its most injurious effects when combined with worry.

The investigation of the family history is of especial value in the study of functional nervous disturbances, as epilepsy, neuralgia, chorea, hysteria, etc., and nervous disturbances symptomatic of diathetic conditions, as gout and syphilis. In all cases it is of value as furnishing hints as to the patient's general condition.

The family history is of interest from still another standpoint. Many times there may be no traces of any disease of this or that particular organ, but the family energy seems to play out at about a particular period of life. The members of the family seem to be endowed by birth with but limited health capital. Information of this kind is practical in enabling us to give the patient the proper information to conserve his physical and mental resources. A danger here creeps in, however. Some of these patients, knowing their family peculiarities, are rendered morbid thereby. Constant dwelling on the subject produces with them so much mental and bodily discomfort as to make them worse off than if they lived regardless of their hypothetical hereditary incapacities.

The history of the patient's illness having been accurately recorded, a physical examination of all the organs should next be undertaken. For accuracy's sake no portion of the body should be overlooked. My own method is one adopted for convenience in the consulting-room rather than because it is scientifically systematic. The parts about the head, the eyes, ears, nose, and mouth are first examined. Then the patient is requested to disrobe, and the thoracic and abdominal viscera are examined. Then I examine the different reflexes. While the patient is dressing, I occupy the time in examining his urine.

If the symptoms complained of relate to alterations in motor and sensory functions, these are examined while the patient is still undressed.

It may seem almost pedantic to the reader to practise the thoroughness above advocated; but I can assure him that oftentimes one comes across, in his physical examinations, the most unexpected conditions, conditions of importance because they have a practical bearing on the illness. Then, too, it must be remembered that nervous symptoms are present—they may even be the predominant conditions—in diseases of every part of the body. Oftentimes when occurring thus, they simulate most closely many diseases whose anatomical substratum is in the central nervous system. It matters not how clearly a given case may coincide with classical descriptions of certain diseases, thorough examination is still necessary. If we neglect it, our patients suffer in health and we in reputation. Let me give some illustrations: Chronic headaches are

often dependent upon refractive errors and diseases of the nasal cavities. Foreign bodies in the ears have produced severe vertigo. Severe and intractable headache has been caused by the constant coughing of an incipient phthisis, the pulmonary symptoms being ignored by the patient. Cases have been ascribed to neurasthenia until the discovery of a double optic neuritis has changed the diagnosis to one of brain tumor. The discovery of albumin or sugar in the urine lets in a flood of light as to the origin of nervous manifestations hitherto unexplainable. And so examples might be multiplied indefinitely.

The methods of physical examination of the various thoracic and abdominal viscera will be described in other portions of this work. The present chapter will, therefore, be confined to a consideration of the examination of the various functions of the nervous system. These are:

- (1) Mental functions.
- (2) Motor functions.
- (3) Sensory functions.

The mental condition of the patient can, as a rule, be determined with tolerable accuracy when obtaining the history of the case.

MOTOR FUNCTION may be disturbed in the direction of excess, diminution and alteration.

Under *excess* we have convulsions, spasms, contractures, tremor, tic, chorea, fibrillation and associated movements.

Under *diminution*, paralysis, which may be localized or general, partial or complete.

Under *alteration* we have disturbances in co-ordination, *e. g.*, ataxia.

Now, to define these various terms:

A *convulsion* is a condition due to excessive discharge of motor impulses from the nerve centres, and characterized by excessive muscular contractions, usually widespread. Convulsions are divided into *tonic* and *clonic*. In the former the muscular contractions are prolonged, and partake of the character of general rigidity; in the latter there are violent relaxations and contractions of muscles producing jerking movements of the convulsed parts. In every case of convulsion inquiries should be made as to the following points:

The time of the first spasm, and the apparently causative conditions.

The duration of the first convulsion, and a full description of its various stages and subsequent phenomena.

The distribution of the convulsive movements.

The frequency of the attacks.

The state of health between the attacks.

The associated symptoms.

A *spasm* is a localized convulsion.

*Choreic movements* are irregular, disorderly movements occurring in



one or more parts of the body, and which bear a certain amount of resemblance to those of a voluntary character. They are typified in chorea.

*Tremor* is a rhythmical to-and-fro movement of small amplitude usually, caused by alternate contraction and relaxation of opposing groups of muscle.

*Fibrillation* is a tremulous contraction of individual muscular fibres producing fibrillary movements, which are felt subjectively or may be visible by the movements of the overlying skin.

*Tic* is a sharp, quick contraction of a muscle, or group of muscles, repeated at varying intervals.

*Associated movements* are movements occurring in part willed to be at rest when some other portion of the body is voluntarily moved.

The physical examination of all symptoms above enumerated is limited to inspection. For full information, careful inquiries must be instituted among the family and attendants.

*Paralysis* is the loss of power to move a portion of the body due to a lesion of the motor nervous apparatus. Inability to move a limb, because of ankylosis or inflammation of joints, local inflammatory conditions or painful affections, must not be regarded as paralysis. The power of contracting the muscles voluntarily is retained, but the patient refrains from exerting this power, because of its inutility or because of the pain it occasions.

In every case of paralysis, the following points should be investigated :

The mode of onset of the paralysis with the associated symptoms.

The distribution of the paralysis.

Its association with rigidities, muscular atrophy, sensory disturbances.

The electrical reactions of the paralyzed muscles.

*Paresis* is a term used to designate paralysis amounting merely to motor weakness.

According to its distribution, paralysis is designated :

*Hemiplegia*, when it involves one lateral half of the body.

*Paraplegia*, when it involves the lower extremities.

*Hemiparaplegia*, when it involves one lower extremity.

*Alternate hemiplegia*, when it involves the arm and leg of one side and the face or eye muscles on the other.

*Monoplegia*, when it is limited to one limb or to a group of muscles.

*Multiple*, when invading two or more disassociated parts.

Paralysis may be detected by inspection of the part while the patient attempts to move it; by dynamometers, and by directing the patient to resist passive movements made by the physician.

Inspection shows the peculiarities in gait known as hemiplegic, paraplegic, and ataxic, all of which will be fully explained in the sections devoted to special diseases. It also shows the inability to perform as extensive movements with the paralyzed parts as with the corresponding part on the opposite side. If the paralysis happens to be bilateral, then one must judge by his knowledge of the standard of the average healthy person. In the case of facial palsy, the peculiar deformity attending the trouble is at once recognizable by inspection.

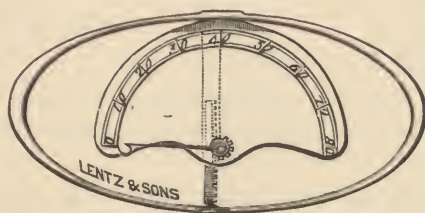


FIG. 12.—DYNAMOMETER.

*Dynamometers* are merely a convenience, not a necessity, in the recognition of loss of power. Their especial value is found in enabling one to make comparisons of the strength of paralyzed muscles from time to time. The one in most common use is that illustrated in the accompanying cut. It is used solely for estimating the grasp of the hand. Many of these instruments are furnished with an inner and outer scale, but this is entirely superfluous. To place any definite value on the readings of dynamometers is liable to lead to error, as these instruments are not accurately calibrated. To say, however, that the patient is capable of exerting sixty degrees of force as shown by the thermometer is perfectly admissible; to say pounds, instead of degrees, is wrong. The readings of dynamometers varying thus, physicians soon learn to appreciate the comparative value of the instruments to which they have been accustomed. There can hardly be said to exist a normal standard of muscular strength. In all cases one must be guided by comparing healthy with diseased parts. I find with the dynamometer which I use, that healthy individuals are capable of registering all the way from fifty-five to one hundred and thirty-five degrees of force—reading by the outer scale. The grasps of the two hands will be found to vary. The right hand is usually the stronger by from ten to fifteen degrees. In ambidextrous individuals there may be no such differences, and in those actually left-handed, the balance of power is in favor of that side. In using the dynamometer, care must be exercised that only the grasping power of the hand is exerted. The patient must not be permitted to lay the hand on some support or to swing the arm in order to gain power.

For the purposes of estimating the strength of the calf muscles, the peronei, the anterior tibial group, the quadriceps extensors, the biceps,

and numerous other muscles, special forms of mechanical contrivances have been designed. They are useful merely for the purposes of making clinical records complete.

The most practical method of estimating loss of power in individual muscles is by resistance to passive motion. For example, to estimate the strength of the biceps, the physician extends the arm at the elbow, while the patient resists.

Paralysis may be also divided according to the seat of the lesion into

- (1) Cerebral,
- (2) Spinal, and
- (3) Peripheral.

*Cerebral paralyses* are of the hemiplegic or monoplegic type, mostly, however, the former. Anæsthesia, when present, is found in the parts paralyzed.

*Spinal paralyses* are of the paraplegic type. Sensory disturbance is frequently the more severe on the side opposite to that in which motor disorder is most marked.

*Peripheral paralyses* are characterized by loss of power in muscles supplied by single nerve trunks, and by the distribution of the anæsthesia in the parts moved by the paralyzed muscles.

**Classification of Paralyses.**—A most excellent classification of paralyses is given by Bastian in his work devoted to the consideration of paralytic affections. The following schema of cerebral and spinal palsies are based largely on his tables. His tabulated differential diagnosis of spinal affections is presented without practical modification.

### Cerebral Paralysis or Lesions of Sudden Onset.

#### I. TRAUMATISM.

(a) *Lacerations of brain substance or compression by depressed bone.*

(b) *Hæmorrhages.*

- (1) Meningeal.
- (2) Intracerebral.

Traumatic hæmorrhages depend generally for their recognition upon the existence of a latent period between the reception of the injury and the appearance of symptoms.

(c) *Meningeal Inflammations.*

These as a rule do not manifest their symptoms until the lapse of two or three days after the injury.

#### II. HÆMORRHAGES.

(a) *Meningeal.*

Usually following blows or falls, syphilitic vascular



disease, arterial degeneration of chronic alcoholism, Bright's disease, and in children from violent paroxysms of coughing.

(b) *Intracerebral*.

Resulting usually from miliary aneurisms, and vascular degenerations from numerous causes, and occurring most frequently after middle life.

III. VASCULAR OCCLUSIONS.

(a) *Thrombosis*.

(1) Arterial.

(i) During convalescence from acute diseases or childbirth.

(ii) From endarteritis (syphilitic or other) or arterial degenerations.

(iii) From feeble and irregular heart action (in combination with either of the above causes).

(2) Venous.

(i) In longitudinal sinus. From general causes, altered blood states, plus a feebly acting heart.

(ii) In lateral sinus. Secondary to inflammation of the scalp and bone, and is either of traumatic origin, or follows caries of the temporal bone.

(b) *Embolism*.

(1) Single.

(2) Multiple.

Paralyses or Lesions of Slow Onset.

I. TUMORS.

II. EXOSTOSES.

III. PACHYMEINGITIS CHRONICA.

I, II, and III, may occur with or without history of traumatism, or with or without evidence of syphilis, tuberculosis, or tumors in other portions of the body.

IV. ABSCESS.

(1) From traumatism.

(2) Suppurative middle-ear disease.

(3) Tuberculosis.

(4) Pyæmic states, secondary to infective lesions elsewhere.

**Paralysis of very Slow Onset.**

- I. DISSEMINATED AND OTHER TYPES OF SCLEROSIS.
- II. MENINGO-ENCEPHALITIS.
- III. SYPHILITIC, ALCOHOLIC, AND SENILE DEGENERATIONS.

**Relative Acuteness or Chronicity of the Several Spinal Affections associated with Paralysis.**

- I. ONSET, ABRUPT.
  - (1) Fractures and dislocation of the vertebræ.
  - (2) Wounds of the spinal cord.
  - (3) Meningeal hæmorrhage.
  - (4) Intra-medullary hæmorrhage.
  - (5) Ischæmia of the lumbar swelling of the cord.
- II. ONSET, ACUTE.
  - (1) Thrombotic softening.
  - (2) Acute myelitis.
  - (3) Acute poliomyelitis anterior.
  - (4) Acute ascending paralysis (?).
  - (5) Caisson disease.
- III. ONSET, SUBACUTE.
  - (1) Intermittent paraplegia.
  - (2) Hysterical paraplegia.
- IV. ONSET, SUBACUTE, OR CHRONIC.
  - (1) Subacute and chronic poliomyelitis anterior.
- V. ONSET, CHRONIC.
  - (1) Cancer of the vertebræ.
  - (2) Scrofulous pachymeningitis from vertebral disease.
- VI. ONSET, VERY CHRONIC.
  - (1) Spastic paraplegia.
  - (2) Ataxic paraplegia.
  - (3) Amyotrophic lateral sclerosis.
  - (4) Locomotor ataxia.
  - (5) Disseminated sclerosis.
  - (6) Diffuse sclerosis (Friedreich's disease).
  - (7) Cervical hypertrophic meningitis.
  - (8) Tumors of the cord and membranes.
  - (9) Syringomyelia.

## TABULAR DIFFERENTIAL DIAGNOSIS OF SPINAL DISEASES ASSOCIATED WITH PARALYSIS.

Group A.	
Spinal Paralysis of Abrupt Onset.	1. Fractures and Dislocations of Vertebræ. 2. Punctured or Gunshot Wounds of Spinal Cord. 3. Concussion of Spinal Cord. 4. Meningeal Hæmorrhage. 5. Intra-Medullary Hæmorrhage. 6. Ischæmia of the Lumbar Swelling of the Cord. 7. Acute Spinal Paralysis. 8. Subacute and Chronic Spinal Paralysis. 9. Acute Ascending Paralysis. 10. Progressive Muscular Atrophy. 11. Pseudo-hypertrophic Paralysis. 12. Amyotrophic Lateral Sclerosis.
Motor Paralysis mostly associated with <i>Muscular</i> <i>Atrophy</i> , and <i>no loss of</i> <i>Sensibility</i> . <i>Pains absent</i> , or very slight.	Obviously the result of Traumatisms.
	More detailed diagnosis as to Seat and Nature of lesion must be based on general principles.
Group B.	Associated with Coldness, and absence of pulsation in arteries, of lower extremities.
	Area of paralysis diminishing after the first few days—leaving one or both legs most frequently paralyzed.
	Onset more gradual, area of paralysis tending to increase from first for some time. Reaction of Degeneration.
	Area of paralysis increasing from the first. No loss of knee-jerk and normal electrical reactions.
	Onset very slow, beginning in upper extremities commonly. Atrophy before paralysis. Muscles react to Faradism.
	Disease of childhood. Paresis; tottering gait, with increased size of calves.
Very Chronic or Chronic.	Disease of adult life. Paresis of upper extremities.
	Twitchings and rigidities of these muscles followed by atrophies. Similar signs in lower extremities with exaggeration of deep reflexes.



<b>Group C.</b>  Chronic Motor Paralyses preceded or associated with <i>Severe Pains</i> in limbs or trunk—with or without evidence of coexisting Vertebral Disease.	13. Cancer of Vertebrae.	{	Secondary to Cancer of Mamma or elsewhere. Disease of middle life or later. Pains very severe. Often rounded prominence of vertebral spines. Legs principally affected. Deep reflexes exaggerated.
	14. Scrofulous Pachymeningitis (with vertebral caries).	{	Patient of scrofulous type, young or early adult age. Pains less severe—often not spontaneous—but in execution of certain movements, or when spine is jarred. Angular curvature often. Legs most commonly affected, with exaggeration of deep reflexes.
	15. Cervical Hypertrophic Pachymeningitis.	{	Disease of adult life. Affecting upper extremities first. Severe pains in them, with hyperaesthesia and afterwards anaesthesia. No evidence of cancer or disease of vertebrae. Followed by paralysis of lower extremities.
	16. Locomotor Ataxy.	{	Lightning pains in lower extremities; absence of knee-jerks. The Argyll-Robertson pupil. Unsteadiness when eyes are closed. Ataxic gait.
<b>Group D.</b>  Diseases in which neither <i>Muscular Atrophies</i> (early) nor <i>Severe Pains</i> are marked features.	17. Tumors and Adventitious Pro-ducts in Spinal Meningitis.	{	By exclusion of other diseases pertaining to Group C.
	18. Softening (Thrombotic).	{	A primary affection. Often sensory as well as motor paralysis. Deep reflexes often exaggerated.
	19. Acute Myelitis.	{	A secondary affection usually.
	20. Disseminated Sclerosis.		
	21. Diffuse Sclerosis (Friedreich's Disease).		
	22. Primary Lateral Sclerosis.		
	23. Intra-Medullary Tumors.		
	24. Toxic Spinal Paralyses.		
	25. Intermittent Paraplegia.		
	26. Hysterical Paraplegia.		
<b>Group D.</b>  Diseases in which neither <i>Muscular Atrophies</i> (early) nor <i>Severe Pains</i> are marked features.	27. Paraplegia Dependent on Idea.		
	28. Reflex Paraplegia.		

These tables apparently give a formidable appearance to the diagnosis of central palsies. Certainly, if an attempt is made to memorize them, they will prove of but little clinical value. Analyzed carefully and applied in the sick-room, they make the subject comparatively easy. Thus a paralysis of sudden onset, due to brain lesion, can be but one of three pathological lesions, viz., traumatism, hæmorrhage or vascular obstruction. A spinal palsy of rapid onset, unattended by pain or disturbance of sensation, can be but two—poliomyelitis and acute ascending paralysis; and the regressive course of the former with the alterations in the electrical reactions and the absent reflexes are sufficient data of differentiation from the latter, which runs a progressive course, has normal electrical reactions and reflexes.

**Hyperkineses or Motor Excitements.**—These include a variety of symptoms, the main characteristic of which is increased or abnormal muscular contractions. The symptoms coming under this head are convulsions, spasms, rigidities, choreic and automatic movements, and tremors. Of these, tremors, choreic movements and automatic movements will be thoroughly considered in subsequent sections of this work. Convulsions also will be treated of under the captions of epilepsy and convulsions, and require but short mention at this time. Clinically, we find convulsions to assume the following types:

- (1) Epileptiform.
- (2) Hysteroid.
- (3) Tetanic.

EPILEPTIFORM CONVULSIONS may occur in:

- (1) Epilepsy.
- (2) Hysteria.
- (3) Organic diseases of the brain.
- (4) From reflex irritation.
- (5) From certain blood states, as uræmia, lithæmia, etc.

It is now an open question whether or not the retention of decomposing food in the gastro-intestinal tract may not give rise to convulsions from toxæmia.

HYSTEROID CONVULSIONS occur in:

- (1) Hysteria.

TETANIC CONVULSIONS occur in

- (1) Tetanus.
- (2) Hysteria.
- (3) Strychnia poisoning.
- (4) Tetany.

The differential diagnosis of these various forms of convulsive movements will be considered under the captions of the diseases above mentioned.

LOCAL SPASMS OCCUR

- (1) Organic diseases of the brain.
- (2) From reflex irritation.
- (3) In hysteria.
- (4) From habit, so-called (habit spasm).
- (5) Cerebral and spinal meningitis.
- (6) In the various occupation neuroses.

Frequent *forms of local spasm* according to distribution are :

- (1) Œsophagismus.
- (2) Spasmodic vomiting.
- (3) Phantom tumor.
- (4) Nervous cough.
- (5) Vaginismus.
- (6) Spasm of the sphincters of rectum and bladder.
- (7) Asthma.
- (8) Facial tic.
- (9) Spasmodic torticollis.
- (10) Rhythmic or functional spasm.

CHOREIC MOVEMENTS are observed in :

- (1) Chorea.
- (2) Hysteria.
- (3) Organic cerebro-spinal diseases.
- (4) Reflex irritation.

CONTRACTURES AND RIGIDITIES are found in :

- (1) Cerebral affections.
- (2) Spinal affections.
- (3) Disease of the peripheral nerves.
- (4) Muscular disorders.
- (5) Functional nervous disease.
- (1) CEREBRAL CONTRACTURES are illustrated in the
  - (A) Immediate, early and late rigidities of hemiplegia.
  - (B) The rigidities of the various forms of cerebral meningitis.
- (2) SPINAL RIGIDITIES AND CONTRACTURES find examples in
  - (A) The late stages of acute poliomyelitis anterior (contractures).
  - (B) Spastic paraplegia and ataxic paraplegia (rigidity).
  - (C) Meningeal irritation.
- (3) Contractures or rigidities in DISEASE OF THE PERIPHERAL NERVOUS SYSTEM are found.
  - (A) In neuritis.
  - (B) In the late stages of incurable peripheral paralyses.
- (4) RIGIDITIES FROM MUSCULAR DISEASE are observed in
  - (A) Thomsen's disease.
- (5) RIGIDITIES AND CONTRACTURES FROM FUNCTIONAL NERVOUS DISORDER are characteristic of some cases of
  - (A) Hysteria.



**Disorders of Sensation.**—These, like the motor functions, are affected in the directions of increase, decrease and alteration of sensibility, known respectively as hyperæsthesia, anæsthesia and paræsthesia. As types of paræsthesia, we have transferred sensations also called allochiria. Delayed sensation may be regarded as a type of anæsthesia.

In an examination of the condition of a given case the sensation should be tested as to

- The tactile sense.
- The temperature sense.
- The pain sense.
- Muscular sensibility.

*Tactile sensibility* is tested by æsthesiometers. This instrument is manufactured in different shapes, the general plan of construction being that of two rather dull points, capable of being moved nearer together or farther apart, at the will of the examiner. The most popular instru-

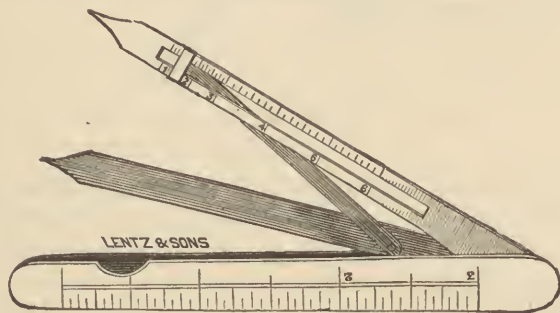


FIG. 13.—VANCE'S ÆSTHESIOMETER.

ment is in the form of a compass (Fig. 14). In using it the patient is blindfolded, so that his vision is precluded from making up for any deficient sensibility. The principle on which it is used depends upon the fact that as the acuteness of tactile sensibility increases, the two points can be distinguished as separate points when more closely approximated. Certain portions of the body are normally more sensitive than others, *e. g.*, the tongue and tips of the fingers. Others, as the back and the thighs, possess comparatively poor sensibility. The following table gives the average distances at which the two points can be distinguished:

- Tip of tongue, 1 mm.
- Tip of fingers, 2 mm.
- Lips, 3 mm.
- Dorsal surface of fingers, 6 mm.
- Tip of nose, 8 mm.
- Forearm, 9 mm.
- Tip of toes, cheeks, eyelids, 12 mm.
- Temple, 13 mm.

Back of hands, 30 mm.

Neck, 35 mm.

Forearm, leg, back of foot, 40 mm.

Back, 60 to 80 mm.

Arm and thigh, 80 mm.

In using the æsthesiometer care must be observed that pressure is exerted equally on the two points, and that they be made to touch the skin simultaneously. They are also more readily distinguished as separate points when side by side at right angles to the long axis of the body or limbs.

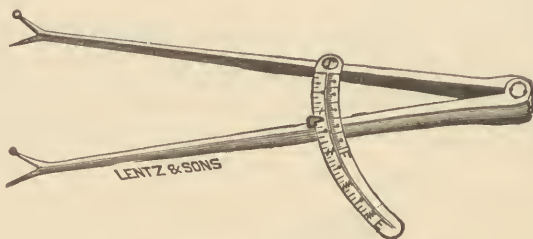


FIG. 14.—CARROLL'S ÆSTHESIOMETER.

Another very useful form of æsthesiometer is one the arms of which are provided with two branches, these being sharp and blunted respectively. It is to be used in determining the patient's ability to recognize sharp and blunted points.

Rumpf has suggested also that tactile sense be tested by writing on the skin with a hard, pointed instrument, the patient's eyes being closed. The more acute sensation is, the smaller are the characters that may be recognized. On the finger-tips the healthy patient should be able to read letters one-fifth of an inch in height.

To test *sense of contact*, a piece of cotton may be drawn lightly over the skin.

To test *sense of locality*, touch any desired portion of the body and direct the patient to touch the corresponding part on the opposite side with an index finger.

To test *temperature sense*, use test-tubes containing hot and cold water. Ordinarily, differences of temperature of three degrees Fahrenheit are appreciable within the limits of 32° F. and 104° F.

*Pressure sense* is tested by determining the differences in weight which can be detected by the skin. Weighted balls are used for this purpose.

The *pain sense* is tested by pricking the parts with a needle or by the application of the faradic brush.

*Muscular sensibility* is determined by the ability of the patient to recognize weights.

The *special sensibility of joints* is determined by moving the limbs in different positions and requiring the patient to duplicate the movements with the opposite limb.

The principal disturbance of sensation observed in practice is its diminution or loss—*anæsthesia*. It may occur as the result of either functional or organic disease. According to its distribution it is known as:

- (1) *Hemianæsthesia*, the loss of sensation being limited to one lateral half of the body.
- (2) *Paranæsthesia*, or loss of sensation of the lower extremities.
- (3) *Monoanæsthesia*, or loss of sensation in one part of the body, as one arm, or one leg, or part of a limb.
- (4) *Alternate hemianæsthesia*, or anæsthesia of one-half of the face and the opposite half of the body.
- (5) *Visceral anæsthesia*.

Hemianæsthesia is always due to a lesion of the sensory tract, between the cortex of the brain and the termination of same in the spinal cord. The lesion may be situated in:

- (a) The spinal cord.
- (b) The medulla oblongata.
- (c) The pons.
- (d) The peduncles.
- (e) The internal capsules.
- (f) The cortex cerebri.

When anæsthesia is due to *disease of the spinal cord*, the sensory loss is situated on the side of the body opposite to the lesion; and if associated with any motor loss, the latter is on the same side of the body.

*Hemianæsthesia from disease of the medulla oblongata* is very rarely noted, for the simple reason that lesions in this situation produce such dangerous symptoms as to result fatally in a very short time.

*Hemianæsthesia from disease of the pons* may be of either the simple or alternate type. When the lesion is situated high in the pons, the anæsthesia is of the alternate variety. The associations of pontine anæsthesias are of the most varied character. They may be combined with motor paralysis of the anæsthetic parts; both sides of the face may be anæsthetic in conjunction with one side of the body; both sides of the face and body may be affected; both sides of the face alone may be anæsthetic; and, lastly, the anæsthesia may be distributed over the one side of the face and both sides of the body.

*Hemianæsthesia from disease of the peduncles* is conceivable, but no cases have been reported.

*Hemianæsthesia from capsular disease* occurs when a lesion involves the posterior third of the hinder limb of the internal capsule, between the lenticular nucleus and the corpus striatum.



Complete *hemianæsthesia from cortical disease* is rare, owing to the fact that the sensory structures in this locality are greatly spread out, so that only an extensive lesion can involve them all.

*Functional or Hysterical Hemianæsthesia*, or, indeed, functional anæsthesias generally, are recognized in the first place by their association with hysterical symptoms, local ischæmia (so that a pin-prick does not produce bleeding), by the shifting of the anæsthesia from one part to another, and by the presence of hyperæsthetic spots in the midst of the anæsthetic area.

The complete hemianæsthesia of organic origin is rarely associated with a high degree of motor palsy.

PARANÆSTHESIA is almost always due to disease of the spinal cord. It may be due to hæmorrhage, inflammation, or sclerosis affecting this organ. Paranæsthesia may exceptionally come on as a result of multiple neuritis.

It may be functional.

MONOANÆSTHESIA is the result either of limited cortical disease or injury or inflammation of single nerve trunks. In the latter case, the anæsthesia follows the rule already mentioned, when the nerve involved is a mixed one, in being especially marked in the parts moved by the paralyzed muscles. The recognition of the fact that the anæsthetic area corresponds to the distribution of a peripheral nerve is a great assistance in diagnosis.

VISCERAL ANÆSTHESIAS are often of hysterical origin, though also organic. They may affect the throat, rectum, bladder and vagina.

*Anæsthesia of the Throat* may occur

- (a) In hysteria.
- (b) In post-diphtheritic paralysis.
- (c) As a part of a general hemianæsthesia.

*Rectal Anæsthesia* is symptomatic in

- (a) Hysteria.
- (b) Locomotor ataxia and other organic spinal affections.
- (c) Widespread cerebral degenerations.

*Bladder and Vaginal Anæsthesias* are found in

- (a) Hysteria.
- (b) Affections of the spinal cord.

**Pain.**—This consideration of pain as a symptom is to be limited to its study only as it occurs in connection with nervous diseases and nervous conditions. Pain accompanying inflammatory and other states manifested by local objective phenomena, as heat and swelling, finds elucidation elsewhere. In practice we find personal susceptibility to pain to vary greatly. Some persons bear with it with great fortitude; suffering greatly and yet making no complaint. Others seem to have the pain sense more or less blunted, and really suffer but little from conditions

which in others would give rise to the greatest agony. Then we find people at the other extreme. A small scratch or the slightest aching calls forth the loudest lamentations. Some magnify their pains by catering to them, as for example, is seen so frequently in the hysterical, whose quiet, placid facial expression contradicts plainly the intensity of their sufferings as so graphically depicted by them in spoken words.

The pains with which we have to do at this time are those arising from the following causes:

- (1) Neuralgia.
- (2) Neuritis.
- (3) Disease of the spinal cord.
- (4) Reflex pains.
- (5) Pains from various toxæmiæ.
- (6) Rheumatic and gouty pains.
- (7) Hysterical pains.
- (8) Anæmia.

*The pains of neuralgia* are characterized by their paroxysmal character in the majority of cases. Still they may be continuous, in which case they are generally dull, and often interrupted by acute exacerbations. They are often associated with the painful points first described by Valleix, and which are situated, speaking in a general way, over points where the nerve emerges from its bony canal, enters a muscle, where its trunk approaches close to the surface of the body, and at the termination of the nerve branches near the surface of the body.

*The pains of neuritis* are limited to the course and distribution of the affected nerve or nerves. Tenderness is experienced along the nerve trunk. The pain may be dull in character, or sharp, shooting and boring. It is greatly aggravated by causes that put the affected nerve on a stretch. They are often aggravated at night, and by changes in weather to cold or damp.

*Pain as accompanying disease of the spinal cord* is very much misunderstood by practitioners generally. It is altogether too common a custom to ascribe many severe spinal pains to this cause, when in reality such cases are of rare occurrence. It must be remembered that backache may occur from a great variety of affections, as disease of the pelvic viscera, gastric and hepatic disorders, neurasthenia, hysteria, disease of the bone and spinal membranes, and chronic inflammation of the fibrous tissues of the spine. Those pains which originate in functional disturbances, as the sensitive spines of neurasthenia, hysteria, etc. (and generally spoken of as spinal irritation), are readily recognized by their tendency to shift from one part of the vertebral column to another; while those arising from organic causes are very likely to be fixed. Organic spinal pains are especially liable to arise from meningitis and disease of the vertebra. In the former the sensitiveness is experienced over the

bones themselves, and in the latter, between the vertebra. They are apt to be especially severe when arising from malignant disease of the bones or meninges. Chronic organic diseases of the cord do not often give rise to severe local back pains.

Peripheral or eccentric pains very frequently occur as symptoms of spinal cord disease. They may arise (1) from irritation of the sensory nerve-roots as they pass through the posterior columns of the cord, through the membranes, or the intervertebral foramina; and (2) from irritation of the sensory conducting tracts in the cord. The eccentric pains are almost always bilateral and symmetrical. They are often of a severe darting character, and their distribution generally indicates the height of the lesion in the cord. The pain from disease of the sensory conducting tracts is generally dull in character, and is very liable to be confounded with the pains of rheumatism, especially as it is very often aggravated by changes to damp or cold weather. A very characteristic pain accompanying spinal cord disease, one, too, that is regarded by many neurologists as positive proof of the existence of organic changes in the cord or its membranes, is the so-called "cincture feeling," or "girdle pain." It is a sensation as of a band about the waist or trunk, and is caused by irritation of the nerve-roots at the lowest portion of the healthy part of the spinal cord. It is found in the different forms of myelitis and meningitis, and in some cases of spinal sclerosis. It sometimes occurs in the lower extremities, then being manifested by a sense of constriction, and is bilateral and symmetrical.

*Reflex pains* are especially interesting, because of their variety and occasional obscurity. Examples are found in the shoulder-pains of liver disease, the backache of gastric ulcer and pelvic disorders, the tenesmus vesicæ from anal fissure, abdominal pain from pericardial hæmorrhage, pain on one side of the face from a bad tooth on the other, in the knee from morbus coxarius, and so one might go on, until a list of great length is presented.

The *toxæmiæ*, which may occasion pain, are malaria, syphilis, diabetes, uræmia, and lithæmia. To mention their existence is to warn against ignoring them in practice. The data for their discovery will be found in the various chapters devoted to the consideration of these subjects *in extenso*.

*Rheumatic and gouty pains* are, as a rule, fixed pains. They are especially liable to be confounded with the pains of traumatism. It must be remembered that very often a sprain of a joint or a traumatism may serve to fix a rheumatic pain to a definite locality and so occasion errors in diagnosis. While rheumatic pains are nearly always aggravated by changes in weather, the pains of neuralgia, neuritis, and organic disease of the cord, are often subject to the same modality. One cannot, then, be too careful in all doubtful cases, to study the case as a whole,



and not to jump too quickly to the conclusion that rheumatism is the trouble.

*Hysterical pains* will be found fully described in the article on hysteria.

*Anæmic pains* are very common, and are recognized by the presence of the characteristic constitutional condition.

Sudden removal of a long-continued pressure will often excite much agonizing pain. The best example of this is found in the severe pains in the legs following rapid delivery.

**Coma and Stupor as Symptoms of Nervous Diseases.**—While in private practice, where it is nearly always possible to obtain a clear history of the case from its very inception, the investigation of coma as a symptom is a comparatively easy matter, in hospital practice one very often encounters insurmountable difficulties, because of the paucity of information at his disposal. The patient is brought in unconscious, having been found in that condition by the police patrol. To determine the cause of the trouble we are obliged to rely upon objective symptoms, and these of the most meagre character. I can recall one puzzling case seen with Dr. T. Hart Smith, in which a man supposed to be in possession of good health was brought home in a stuporous state, gradually lapsed into coma, and died. His breath had the odor of laudanum, as did the stomach washings, but aside from the stupor his symptoms were not those of opium poisoning, and he had taken only a medicinal dose of that drug. An autopsy showed an old syphilitic kidney, with all other viscera healthy. And so the case remained a mystery. A man returning from the Naval Review in New York was taken with drowsiness on the train; by the time he reached Philadelphia he was stupid. He was then removed to the Hahnemann Hospital. He lapsed into coma, and died three days later. The autopsy showed tubercular meningitis, and yet he had believed himself to be in perfect health up to the beginning of the comatose state.

Coma is a state of insensibility or unconsciousness from which the patient can be but partially or not at all aroused.

STUPOR consists of a state of apathy or drowsiness.

Coma and stupor may be regarded as different degrees of intensity of the same symptom.

Coma is symptomatic in:

- (1) Cerebral traumatism.
- (2) Acute alcoholism.
- (3) Narcotic poisoning, *e. g.*, opium and its alkaloids.
- (4) Sunstroke.
- (5) Cerebral hæmorrhage.
- (6) Cerebral embolism and thrombosis.
- (7) Epilepsy.

- (8) Cerebral tumor.
- (9) Cerebral abscess.
- (10) Cerebral meningitis.
- (11) General paralysis of the insane.
- (12) Uræmia.
- (13) Diabetes mellitus.
- (14) Yellow atrophy of the liver.
- (15) Syphilis.
- (16) Acute infectious diseases of the foudroyant type.

No subject concerning the question of coma is of greater practical relative importance than its relation to traumatism. The text-books teach that in the absence of objective evidence pointing to fracture of the skull, such unconsciousness is due to concussion of the brain. This is a pernicious doctrine, fraught with such danger to the injured as to deserve the designation of inhumane. The advanced neurological and surgical sentiment of the day antagonizes the very existence of concussion of the brain and spine as actual entities. Careful investigation will show in every case laceration of brain substance, fractured skull, meningeal injury, or hæmorrhage. If the term cerebral concussion is to be retained, it should be limited to those cases in which unconsciousness lasts but a short time, an hour or two at the most, after the injury. Even in these it must be admitted that probably the lesion is of too slight a character to be discernible by our means of research, rather than of the mysterious character attributed to concussion. Head injuries with coma or stupor should be treated with the most profound respect. Owing to the very slight danger attendant upon antiseptic incisions, it is better by far to at once explore the seat of injury most carefully.

The coma of acute alcoholism is rarely, if ever, so deep but that the patient can be partially aroused therefrom by shaking or loud calling.

The coma of narcotic poisoning (opium) is attended by contracted pupils. There is no evidence of hemiplegia.

The comas of sunstroke, cerebral hæmorrhage, embolism, tumor, abscess, and general paralysis of the insane, will all receive attention in their respective articles.

**The Reflexes.**—An examination of the various reflexes is a very important routine measure in all cases.

The reflexes are divided into

- (1) The *superficial* or *cutaneous reflexes*, and
- (2) The *deep* or *tendinous reflexes*.

The superficial reflexes are certain reflex movements produced in response to irritation excited in definite areas of the body. The value of the reflexes lies in the fact that their presence proves conclusively that the integrity of the corresponding reflex arc is intact. This reflex arc, as illustrated in the accompanying figure, consists of the motor and sen-

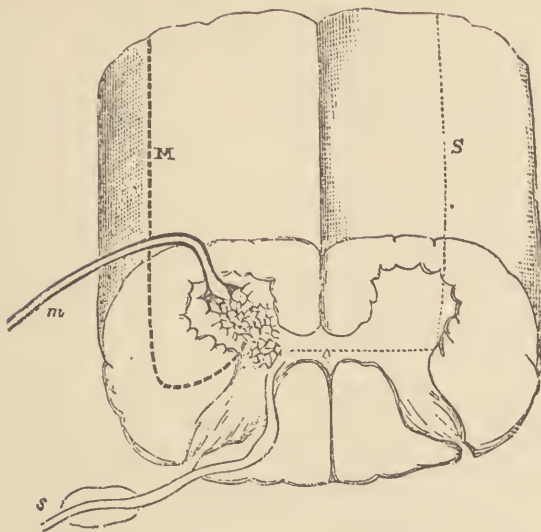


FIG. 15.—DIAGRAM OF A REFLEX LOOP.

sory fibres of the peripheral nerve, the anterior and posterior nerve-roots, the anterior cornua, and the posterior root zones. Any solution of continuity in this path destroys the integrity of the arc, and the reflex movement is prevented when the proper irritation is applied.

The *superficial reflexes* are as follows: Plantar, gluteal, cremaster, abdominal, epigastric, scapular, and erector spinæ.

The *plantar reflex* is excited by irritation of the sole of the foot, which produces movements of the foot and toes. Its presence depends upon the integrity of the reflex arcs from the fifth lumbar to the second sacral segments.

The *gluteal reflex* is produced by irritation of the skin over the buttock, which produces contraction of the glutei. It depends upon the integrity of the arcs from the fourth and fifth lumbar segments.

The *cremaster reflex* is produced by irritation of the skin on the inside of the thighs. The resulting movement is sudden retraction of testicle on corresponding side. It depends upon the integrity of the first to the third lumbar segments. Care must be taken in examining this reflex, not to confuse with it the vermicular movements of the dartos. When well-marked, the cremaster reflex may be excited by slight irritation of the outer or anterior surface of the thigh.

The *abdominal reflex* is produced by irritation of the skin over the side of the abdomen on a line perpendicular to the nipple. The resulting movement is a contraction of the rectus muscle of the corresponding side. It depends upon the integrity of the eighth to the twelfth dorsal segments.

The *epigastric reflex* is produced by irritation of the side of thorax on the line of the nipple. The resulting movement is a dimpling of the



epigastrium. Its existence depends upon the integrity of the reflex loops from the fourth to the eighth dorsal segments.

The *scapular reflex* is produced by irritation of the skin between the scapulæ. The resulting movement is contraction of certain of the scapular muscles. It depends upon the integrity of the reflex loops of the fifth cervical to the first dorsal segments.

The *erector spinæ reflex* is produced by irritation of the skin on either side of the spinous processes.

The tendon reflexes of practical value are the *knee-jerk*, the *ankle-clonus*, and the *triceps-jerk*. A tendon reflex phenomenon is possible wherever there is a tendon that can be put on the stretch and a blow applied to it. Of the tendon reflexes the one known as the knee-jerk is the most important. A proper understanding of the methods by which it is elicited is in order. The method most commonly in use is the following: The patient should be seated. The knees should be bared. Then the leg to be examined should be thrown over its fellow, and allowed to hang as if lifeless, so that no involuntary rigidity shall be permitted to interfere with the reaction. Then with the outer edge of the hand, or with a percussion hammer, or the edge of the ear-piece of



FIG. 16.—TAYLOR'S PERCUSSION HAMMER.

a stethoscope, a smart blow should be applied to the ligamentum patellæ, just below its insertion into the patella; when in health, a vigorous contraction of the quadriceps extensor ensues, and the foot is jerked suddenly upwards. In applying the test, baring of the knees is absolutely necessary for the sake of accuracy in observation; especially is this so in cases in which the blow applied through the clothing has failed to elicit a response. It cannot be conclusively stated that the patellar reflex is absent in any given case, unless this be done. No less important is the injunction that the limb examined be held perfectly limp and lifeless. Frequently have I seen cases in which the patellar tendon reflex had been declared to be absent, simply because the precaution of securing perfect relaxation of the limb had been neglected. There are cases in which this is a difficult matter, owing to the inability of the patient to fix his attention away from the operator's movements. In such it has been my custom to adopt the device first suggested by Jendrassik, to semi-flex the fingers, hook them together and exert strong traction. While this is being done, the blow is applied to the

ligamentum patellæ as before, when, if the knee-jerk be present, it is readily elicited. Any other voluntary movement made by the patient will secure the same object. Thus asking the patient to count aloud, or to look into vacancy and wink rapidly, and while the physician applies the blow to the ligamentum patellæ, will secure a response as readily as by the method of Jendrassik.

In very stout persons the above method of applying the patellar reflex is impracticable, owing to their inability to sit with legs crossed. In such cases the patient should be directed to sit on the edge of a table or desk with the legs dangling over. A blow applied to the ligamentum patellæ, as in the previously described method, produces the knee-jerk.

Cases will frequently occur in the practice of all of us in which the patient's illness is of such a nature as to make it impossible to get him out of bed to apply one of the above-described tests. We may then resort to the following method, first suggested by Dr. Angel Mooney. The patient lying on the back, the thigh is flexed at right angles with the body, and the knee at nearly a right angle with the thigh; the foot is then supported loosely in the palm of the operator's hand, as in a stirrup, care being taken that the patient exert no effort to maintain the limb in this position. A blow is then applied to the ligamentum patellæ, as in the other methods of examination; the knee-jerk then takes place.

The *ankle-clonus* is produced as follows: The examiner takes the patient's leg, supporting the heel on his knees, and the popliteal space with the left hand, observing care that the limb is slightly flexed at the knee-joint. Then with the right hand applied to the ball of the foot, that member is pushed suddenly upward, maintaining the foot in dorsal flexion, and putting the tendo-Achilles on a stretch. If the ankle-clonus is present, there appears a rhythmical movement of the foot, which, in case the phenomenon is at all well marked, continues for several minutes or until the pressure is removed. Unlike the patellar reflex, the ankle-clonus is a pathological condition.

The *triceps-jerk* is produced as follows: The patient sits with arm flexed at somewhat of an obtuse angle at the elbow, the forearm resting lightly on the lap. A blow, applied by means of the percussion hammer (Fig. 16) to the triceps tendon, produces the elbow-jerk.

The reflexes are absent in any lesion impairing the integrity of the reflex above described. We therefore find them absent in:

Nerve injuries.

Neuritic paralysis.

Lesions of the spinal nerve-roots.

Acute anterior poliomyelitis.

The tendon reflexes are absent in the above, and in addition in:

Locomotor ataxia.

Cerebellar disease, sometimes.

Meningitis, sometimes.

In the earliest stages of apoplectic extravasations.

The cutaneous reflexes are absent or diminished and the tendon reflexes exaggerated in :

Cerebral lesions.

Both, deep and superficial reflexes are exaggerated in :

Spinal cord lesions involving the lateral tracts of the cord, hence in spastic paraplegia, ataxic paraplegia, disseminated sclerosis, some cases of tumor of the cord, and pachymeningitis from Pott's disease.

In neurasthenic and hysterical conditions.

Exhausting diseases.

**Electro-Diagnosis.**—An investigation into the reactions of the paralyzed muscles to both the galvanic and faradic currents, forms a very important part of the examination of many cases ; indeed, in quite a number of them it is absolutely essential to a correct conclusion. For electro-diagnostic purposes, both forms of battery are needed. The faradic machine should be one capable of furnishing a smooth, steady current, and it should be provided with a device for automatic slow interruptions of the current. The galvanic battery should be one possessing an electro-motive force of not less than fifty volts, and it must be provided with a commutator for reversing the poles, a rheostat for increasing or decreasing the strength of the current, and a milliamperemeter, for measuring the amount of current in use. One can, of course, get along without either a rheostat or a milliamperemeter, just as physicians were obliged to do before these instruments were adapted to medical use ; but it is decidedly inconvenient to vary the strength of a current by altering the number of cells in the circuit, to say nothing of the unpleasantness of this procedure to the patient, and the absence of a milliamperemeter makes our observations decidedly lacking in accuracy. One has only to become used to these instruments to become convinced of the inaccuracy of all his previous observations. Of the portable faradic batteries, I have used with satisfaction those manufactured by Flemming of Philadelphia, and the Chloride of Silver Battery Company, of Baltimore. Both instruments have given entire satisfaction. Of portable galvanic batteries, I now use only the chloride of silver battery of fifty cells, provided with a Willms current regulator and a milliamperemeter. For office purposes, I have used for a number of years one of Flemming's wall cabinets, provided with a Dubois-Reymond coil for the faradic current, and a Massey current regulator and Flemming milliamperemeter. The current is supplied by sixty Law cells. Such an elaborate arrangement for a stationary battery is not necessary, however, as any physician with mechanical ingenuity can manufacture his own cells, and by purchasing the



current regulator and the milliamperemeter, can obtain as good results as he can with an apparatus of a more finished construction. The only objection that can possibly be offered to the chloride of silver battery is that of expense, and this is more apparent than real. After the first outlay the cost of maintenance is practically *nil*, while the cleanliness of the instrument and the readiness with which it may be used, give it a great advantage in the way of economy over the many varieties of acid battery.

The application of either the faradic or the galvanic currents to healthy nerves and muscles produces muscular contractions. In the case of the nerve, the contractions are exhibited in all the muscles to which it conducts motor power. In the case of the muscles, the contractions occur only in those directly stimulated. With the faradic current, the muscular contractions are of a tetanic character and continue as long as the current passes, or until the irritability of nerves and muscles is exhausted.

The reactions to the galvanic current require longer explanation. In the first place, muscular contractions take place only at moments of making and breaking the circuit, excepting when very powerful currents are employed, when a tetanic contraction appears. Muscular contractions are more readily obtained by closing the current and applying the negative pole. This is called the cathodal closing contraction, and is designated as the  $\text{CaClC}$ . Muscular contractions also take place on breaking the circuit with the negative pole applied, but a much greater strength of current is necessary to effect this result. This is called the cathodal opening contraction and is symbolized,  $\text{CaOC}$ . The contraction at the positive pole on breaking the circuit is somewhat stronger than that on completing the same, and these are designated respectively, anodal opening and anodal closing contractions, and their symbols are  $\text{AnOC}$  and  $\text{AnClC}$ . We have then, as the order of irritability of muscles in response to galvanism, the following :

- (1)  $\text{CaClC}$ .
- (2)  $\text{AnOC}$ .
- (3)  $\text{AnClC}$ .
- (4)  $\text{CaOC}$ .

Disease alters the electrical reactions of paralyzed muscles, oftentimes in a characteristic manner. In certain forms of paralysis, by reason of long-continued disuse of the affected part, the electrical irritability of the paralyzed muscles becomes greatly diminished alike to both galvanic and faradic currents; in others, the phenomena known as the reaction of degeneration present themselves; and in still another class of cases no departure from the normal is observed. This last-mentioned fact needs to be emphasized, because it is a too prevalent idea that actual paralysis dependent upon organic changes must give evidence of its genuineness in the electrical reactions of the paralyzed muscles. This is a mistake;

this warning should lead the examiner not to be too ready in deciding that such cases are examples of malingering.

A certain routine in the electro-diagnosis of paralytic affections is advisable to secure accuracy in results. At the outset it must be remembered that the muscles and nerves in healthy persons do not respond to electrical stimulation with equal vigor to the same strength of current. There being, therefore, no arbitrary health standard in this respect to determine what is normal for the patient under examination, one must first examine the electrical reactions of the muscles on the healthy side. If the paralysis happen to be bilateral, then certain normal muscles must be examined, their irritability compared with that of the average individual, and a standard for the case in hand accordingly determined.

When preparing the apparatus for the examination, every attention must be paid to details. The electrodes must be properly covered and moistened with warm or salt water, and the battery known to be in perfect order, precautions altogether too frequently neglected, self-evident though they be. One electrode is used over the muscle or nerve to be tested, and the other is applied over some indifferent portion of the body, one comparatively free from motor nerves and muscles being best. The point usually selected for the latter is the sternum. This electrode should be a large flat one. For purposes of description it may be referred to as the "silent electrode." The other electrode may be in size from one-quarter of an inch to one inch in diameter; it is termed the active electrode. The greater the operator's skill, the smaller will be the active electrode with which he can work successfully.

The instruments being ready, the examiner should first test the irritability of nerves and muscles to the faradic current. Let him use the negative electrode as the active one. He must begin as already stated by determining the health standard. Having discovered the strength of current sufficient to produce contractions in healthy nerve or muscle, then he should study the effect of the same current applied to the corresponding point on the diseased side, and note the result. In every case it is important that he pay strict attention to the correct placing of the active electrode over the motor points of the nerve or muscle to be tested. The motor point is usually over the site of entrance of a nerve into a muscle, or at a spot where the nerve comes nearest the cutaneous surface. The accompanying cuts give the motor points of the most important nerves and muscles, and are important from a therapeutic as well as from a diagnostic standpoint, in the electrical treatment of paralysis. In the case of the faradic examination it is only important to note diminished, absent, or increased muscular contractions. In the case of the galvanic current, other details of observation must be attended to. The electrodes are applied as before. The active electrode must be attached to an interrupting handle, so that the

effects of sudden making and breaking of the circuit may be watched. It is best to begin with the negative as the active electrode, and the minimum current capable of producing muscular contractions at making and breaking of the circuit, determined. The character of the muscular contractions must also be observed. In health, the muscles respond with a short, quick jerk. Having determined the electro-muscular irritability to the negative pole, the same should be investigated with the positive pole, and the results noted. Departures from the galvanic reactions in health may be noted in several directions. In the first place, the irritability of the nerves may be diminished or increased, in direct proportion to the alterations in faradic contractility. The muscles themselves may be similarly affected. These changes are called quantitative alterations. In the second place we may find important changes in the character of the reactions. These are known as qualitative alterations. The galvanic irritability of the muscles may be increased, while the faradic is diminished or even destroyed. The resulting muscular contractions are of a slow wavy character, and not short and quick as in health; and there are important changes in the polar reactions. Thus, instead of the cathodal closing contraction being the most marked, we find the anodal opening or anodal closing contraction the strongest. For purposes of record we may use the symbols already given, and designate their relations by the use of the crescendo, diminuendo, or equality signs. Thus  $\text{CaClC} > \text{AnOC}$  would indicate cathodal closing contraction stronger than anodal opening contraction. Reversing the sign, making it a diminuendo in other words, and we indicate the anodal opening contraction as the stronger.

While definite rules respecting the alterations in the polar reactions have been formulated, they are by no means reliable clinically, as their significance can hardly be said to have as yet been determined. These qualitative alterations in galvanic response, together with diminished or absent faradic irritability, constitute the reaction of degeneration. For all practical purposes in actual practice, it is necessary only to discover the diminished faradic and increased galvanic response, and the alterations in the character of the muscular contractions. In many cases in which the faradic irritability is so slightly altered as to be almost imperceptible, the altered galvanic response becomes of the greatest importance.

Departures from the characteristic reaction of degeneration as above described may be observed under certain circumstances. One is known as the chronic form and is characterized by diminution of faradic and galvanic irritability of nerves and muscles alike, with the qualitative galvanic changes above described. The other is called the "mixed form." It is characterized by slight or marked changes in the muscle irritability, while the nerves react normally or nearly so. The altera-



tions in the muscle reactions consist of increased or diminished galvanic irritability and changed polar reactions. This condition is observed in cases in which some of the muscular fibres are degenerated, while others are normal, as in some cases of peripheral paralysis and progressive muscular atrophy.

The reaction of degeneration in its typical form has a well-defined course. At about a period ranging from two or three days to as many weeks, there appears the diminished faradic irritability, which progresses more or less rapidly to complete destruction according to the severity of the case. At the same time the galvanic irritability of the nerves diminishes. In about from one to two weeks the galvanic irritability of the muscles increases as above stated, and reaches its height in about a month, when it too begins to diminish. In marked cases it may go on to complete abolition. At periods ranging from two to six months restoration takes place in a measure in some cases, this of course being governed by the severity of the primary lesion.

The reaction of degeneration is of diagnostic value, as it indicates certainly a lesion affecting a motor nerve or its nuclear cells. Hence we find it present in acute poliomyelitis, nerve injuries and inflammations, and in degenerations of the nuclei of the cranial nerves. It is found in its chronic form in progressive muscular atrophy, and amyotrophic lateral sclerosis.

Simple diminished electrical irritability without qualitative changes is found in cerebral palsies after the paralysis has lasted three or four months, in certain spinal cord diseases, as amyotrophic lateral sclerosis, and after slight nerve injuries and inflammations, and in certain muscular conditions, as arthritic muscular atrophy, atrophy from disuse and myopathic atrophy.

Increased electrical irritability without qualitative changes is observed in the early stages of hemiplegia and of acute myelitis, progressive muscular atrophy, chorea, locomotor ataxia, and Thomsen's disease. In the last named a peculiar condition is sometimes observed. It is called the myotonic reaction, and it consists of increased faradic contractility with prolonged contraction after the current ceases, and in excessive galvanic irritability when the circuit is closed with sluggish and wavy contractions.

The many other symptoms, significant of nervous disease and nervous complications of other disorders, might well be considered with advantage at this time. Inasmuch, however, as they will be fully treated in other portions of this work, their study here is unnecessary.

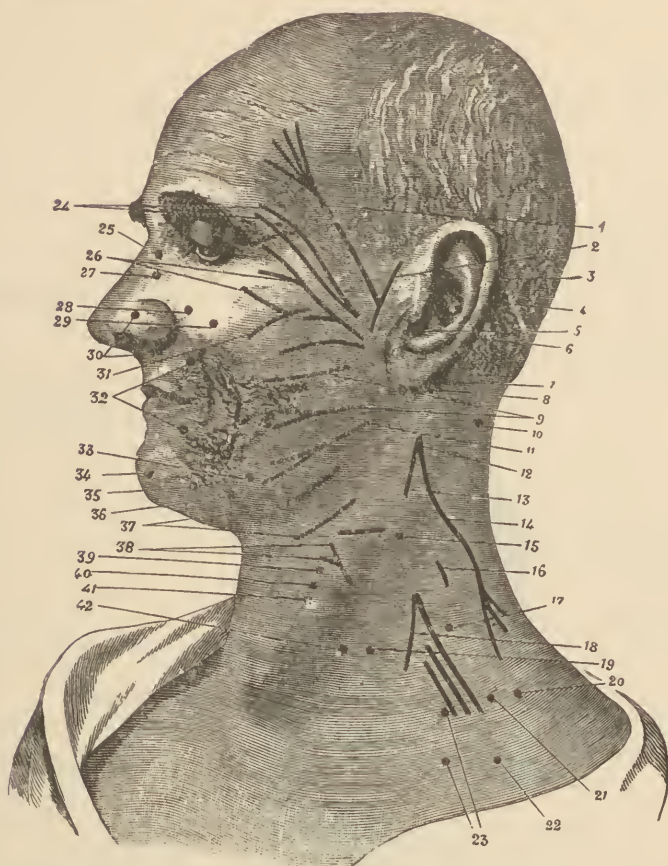


FIG. 17.—MOTOR POINTS OF HEAD AND NECK (ZIEMSEN).

- |  |   |
|--|---|
| 1. Frontalis muscles.                                  | 22. Branch of brachial plexus (musculo-cutaneous and part of median). |
| 2. Attrahens and attollens auriculam muscles.          | 23. Anterior thoracic nerve (pectoral muscles).                       |
| 3. Retrahens and attollens auriculam muscles.          | 24. Corrugator supercilii muscles.                                    |
| 4. Occipitalis muscle.                                 | 25. Compressor nasi and pyramidalis nasi muscles.                     |
| 5. Facial nerve.                                       | 26. Orbicularis palpebrarum muscle.                                   |
| 6. Posterior auricular branch of facial nerve.         | 27. Levator labii superioris alæque nasi muscle.                      |
| 7. Stylohyoid muscle.                                  | 28. Levator labii superior muscle.                                    |
| 8. Digastric muscle.                                   | 29. Zygomaticus minor muscles.  |
| 9. Buccal branch of facial nerve.                      | 30. Dilator uris.   |
| 10. Splenius capitis muscle.                           | 31. Zygomaticus major.  |
| 11. Subcutaneous branches of inferior maxillary nerve. | 32. Orbicularis oris.   |
| 12. External branch of spinal accessory nerve.         | 33. Branch to triangularis and levator menti muscles.                 |
| 13. Sterno-mastoid muscle.                             | 34. Levator menti muscle.   |
| 14. Cucullaris muscle.                                 | 35. Quadratus menti muscle.   |
| 15. Sterno-mastoid muscle.                             | 36. Triangularis menti muscles.                                       |
| 16. Levator angulæ scapulæ muscle.                     | 37. Cervical branch of facial nerve.                                  |
| 17. Posterior thoracic nerve.                          | 38. Branch to platysma muscle.  |
| 18. Phrenic nerve.                                     | 39. Sternohyoid muscle.   |
| 19. Omohyoid muscle.                                   | 40. Omohyoid muscle.  |
| 20. Nerve to serratus magnus muscle.                   | 41. Sternothyroid muscle.   |
| 21. Axillary nerve.                                    | 42. Sternohyoid muscle.   |

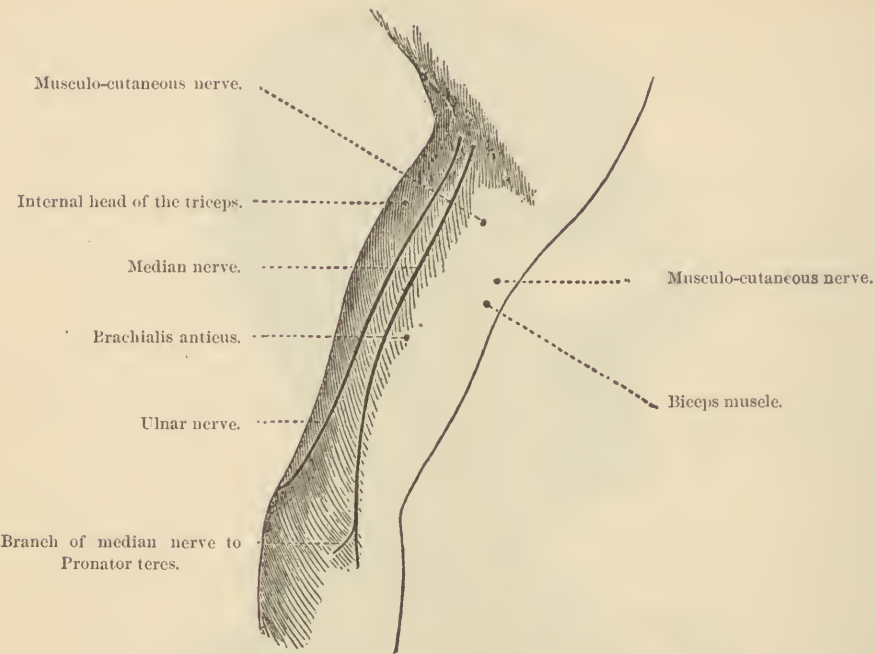


FIG. 18.—MOTOR POINTS OF THE ARM (ZIEMSEN).

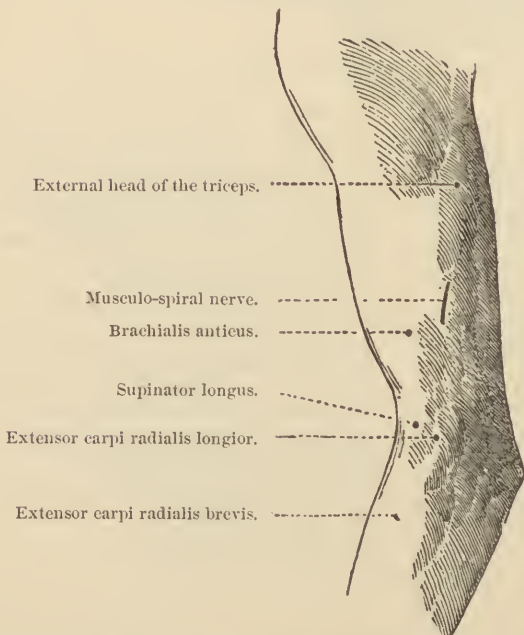


FIG. 19.—MOTOR POINTS OF POSTERIOR SURFACE OF THE ARM (ZIEMSEN).



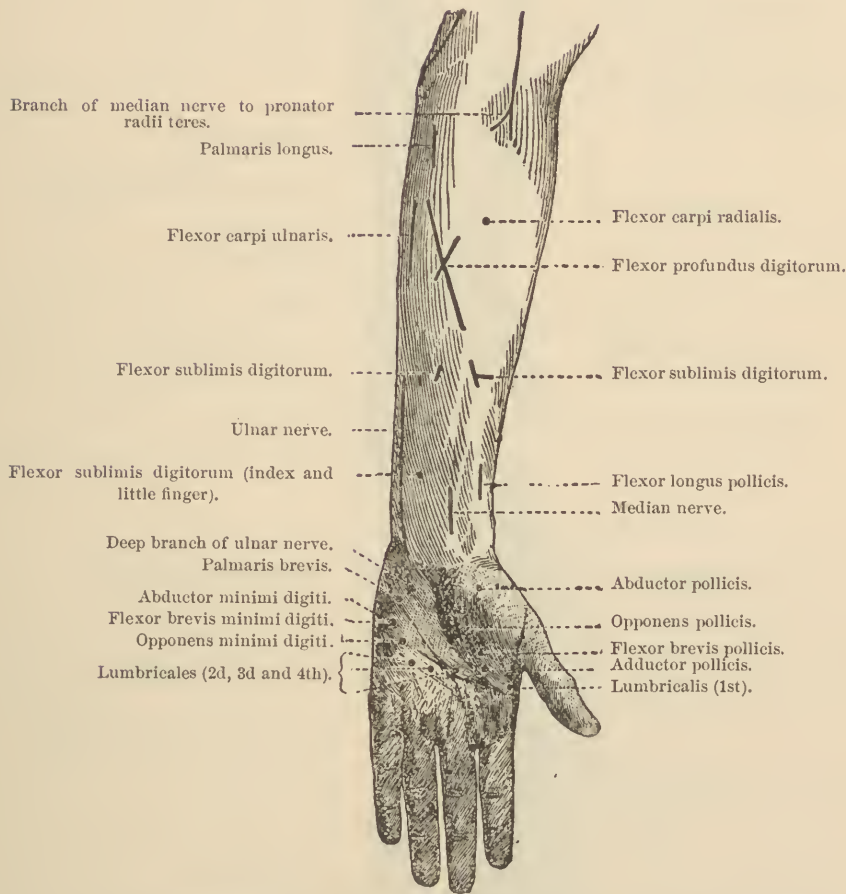


FIG. 20.—MOTOR POINTS OF FLEXOR ASPECT OF FOREARM (ZIEMSEN).

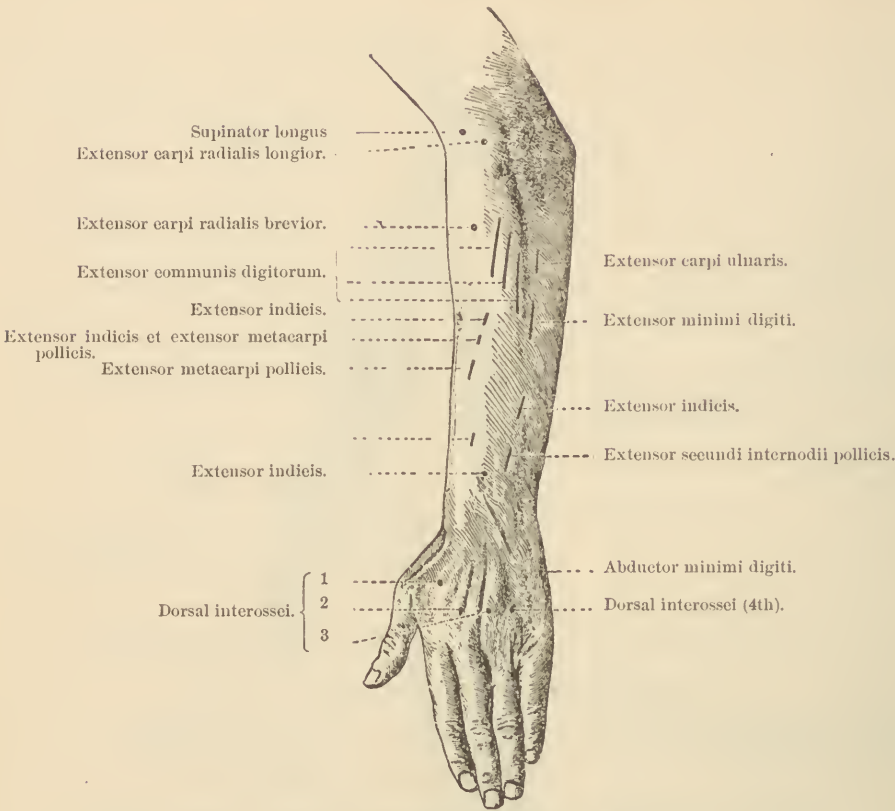


FIG. 21.—MOTOR POINTS OF EXTENSOR ASPECT OF FOREARM (ZIEMSEN).

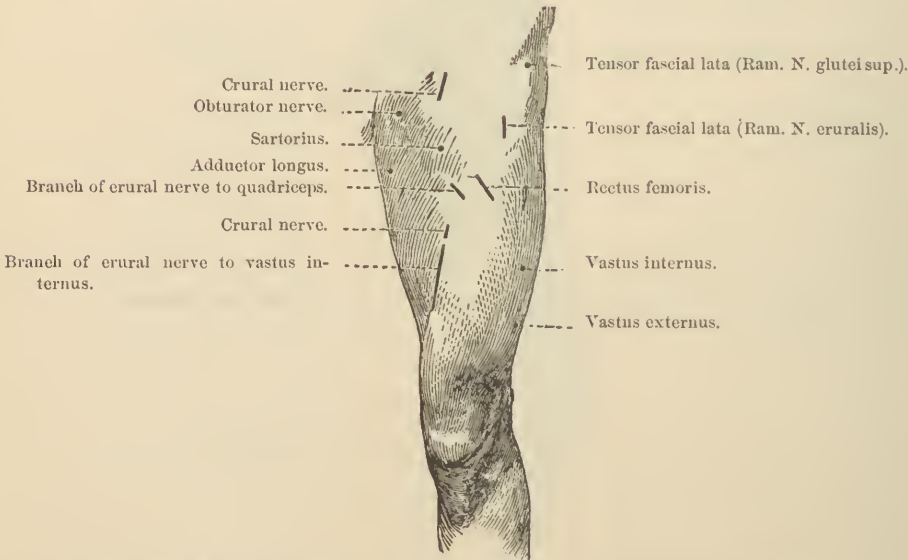


FIG. 22.—MOTOR POINTS OF ANTERIOR SURFACE OF THIGH (ZIEMSEN).

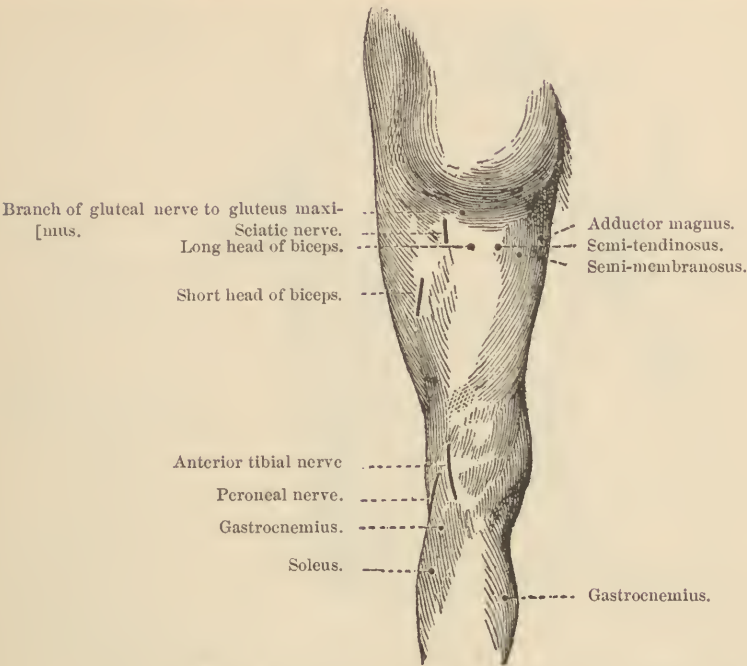


FIG. 23.—MOTOR POINTS OF POSTERIOR SURFACE OF THIGH (ZIEMSEN).



FIG. 24.—MOTOR POINTS OF OUTER SURFACE OF LEG (ZIEMSEN).



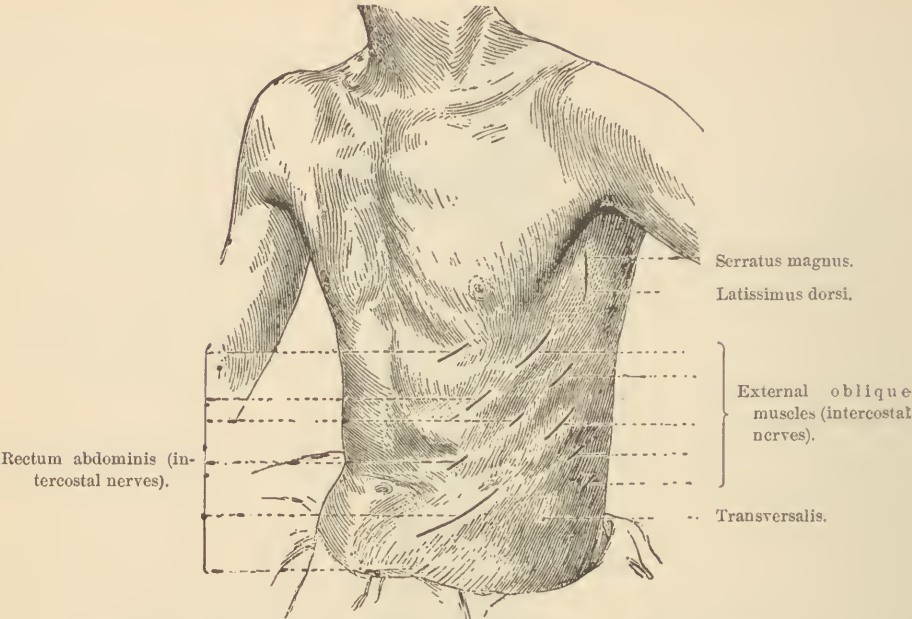


FIG. 25.—MOTOR POINTS OF ANTERIOR SURFACE OF TRUNK (ZIEMSEN).

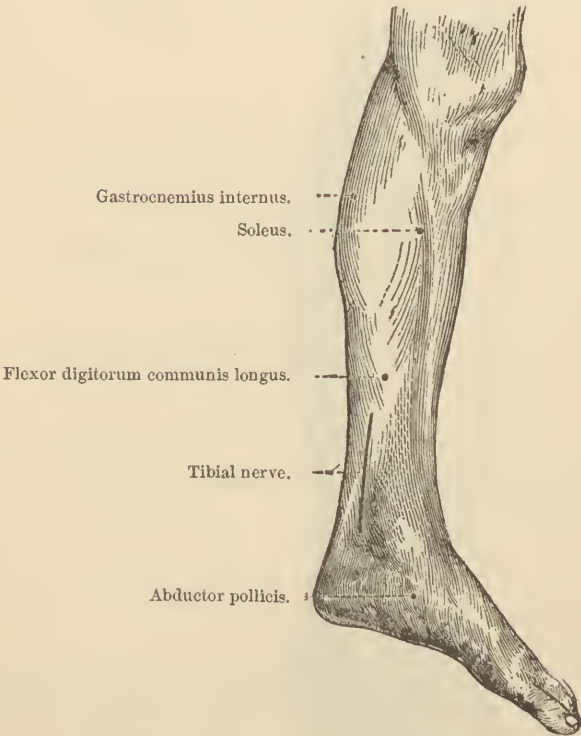


FIG. 26.—MOTOR POINTS OF INNER SURFACE OF LEG (ZIEMSEN).

**Diagnosis.**—The diagnosis in the case of nervous diseases endeavors to accomplish two objects: (1) The determination of the nature of the lesion, *i.e.*, the pathological diagnosis; and (2) the localization of the lesion. From a standpoint of treatment, as well as of prognosis, the pathological diagnosis is the more important. It is also the more difficult. It can only be successfully made, when the physician is in possession of a thorough knowledge of all the essentials of pathology and clinical medicine. The difficulties arise from the complications and lesions, and our inability as yet to recognize certain morbid changes which produce no known special phenomena of their own. We may be able to diagnose a case as one of tumor; but we are unable to state positively its nature, although there are certain facts which enable us to guess the same with some accuracy; but we can never determine short of actual inspection the secondary changes produced by the tumor.

The differentiation of functional and organic disease requires a thorough knowledge of the entire domain of medicine. In the first place it is necessary to be able to exclude all possibility of organic diseases of the nervous system; in the second, to exclude organic diseases of other portions of the body; and in the third place, to recognize the causes which give rise to functional affections.

### LOCALIZATION OF SPINAL CORD LESIONS.

The functions of the different systems of fibres of the spinal cord are not yet by any means fully understood. It is probably not incorrect to say that these are fibre systems, whose very existence is not even suspected. Thus it is while we possess considerable knowledge of practical value pertaining to spinal localization, the subject is yet in its infancy. The present article will be confined to the study of lesions of the more prominent and better understood parts of the organ.

In the localization of a lesion, it is necessary to decide as to its height in the spinal cord, and as to the system it involves in the affected segment. The former is based on a knowledge of the functions of the several spinal segments; the latter, on the physiology of the different system fibres and groups of nerve-cells.

For determining the height of a lesion, the following table modified from Starr is all sufficient:

SEGMENT OF CORD.	NERVES.	MUSCLES.	SENSORY AREAS.	REFLEXES.
Second and third cervical.	Occipitalis, major and minor. Auricularis mag. Superficialis colli. Supra-clavicular.	Sterno-mastoid. Trapezius. Scaleni and neck muscles. <i>Diaphragm.</i>	Neck and back of head.	
Fourth cervical.	Supra-clavicular. Circumflex. Musculo-spiral. Musculo-cutaneous.	<i>Diaphragm.</i> Supra- and infra-spiuatus. Deltoid. Supinator longus. Rhomboidei.	Neck. Superior surface of shoulder. Outer surface of arm.	Cilio-spinal (4th cervical to 2d dorsal).
Fifth cervical.	Supra-clavicular. Circumflex. External cutaneous. Internal cutaneous Posterior spinal branches.	Deltoid. Biceps and coracobrachialis. Rhomboidei. Deep muscles of the shoulder-blade. Pectoralis (clavicular part). Teres minor. Serratus magnus. Brachialis anticus. Supinator longus and brevis.	Back of shoulder and arm. Outer side of arm and forearm.	Scapular (5th cervical to 1st dorsal). Biceps jerk. Supinator jerk.
Sixth cervical.	External and internal cutaneous. Radial.	Pectoralis (clavicular part). Subscapular. Serratus magnus. Biceps. Brachialis anticus. Triceps. Extensors of wrist and fingers. Pronators.	Outer side and front of forearm. Back of hand (radial distribution).	Elbow jerk. Wrist jerk.
Seventh cervical.	External and internal cutaneous. Radial. Median. Posterior spinal branches.	Triceps (long head). Extensors of wrist and fingers. Pronators. Flexus of wrist. Subscapular. Pectoralis (costal portion). Serratus magnus. Latissimus dorsi. Teres major.	Radial and medial distribution.	Palmar (7th cervical to 1st dorsal).



SEGMENT OF CORD.	NERVES.	MUSCLES.	SENSORY AREAS.	REFLEXES.
Eighth cervical.	Internal cutaneous ulnar.	Triceps (long head). Flexors of wrist and fingers. Small muscles of the hand.	Inner side of arm and forearm. Ulnar area of hand.	
First dorsal.	Internal cutaneous nerve of Wrisberg.	Extensors of thumb. Small muscles of the hand. Thenar and hypothenar muscles.	Inner side of arm and forearm.	
Second dorsal.	Intercosto - humeral.		Inner side of arm, near axilla.	
Second to twelfth dorsal.	Intercostals and dorsal posterior nerves.	Muscles of back and abdomen. Erectores spinæ.	Skin of back and upper gluteal region, and of breast and abdomen, in bands running downward and forward.	Epigastric (4th to 8th dorsal). Abdominal (8th to 12th dorsal).
First lumbar.	Ilio-hypogastric. Ilio-inguinal.	Ilio-psoas. Sartorius. Rectus.	Skin over groin and front of scrotum.	Cremaster (1st to 3d lumbar.)
Second lumbar.	Genito-crural. External cutaneous.	Ilio-psoas. Sartorius. Quadriceps femoris.	Outer surface of thigh.	
Third lumbar.	Anterior crural. Internal cutaneous. Long saphenous. Obturator.	Quadriceps femoris. Anterior part of biceps. Inner rotators of thigh. Adductors of thigh.	Anterior surface of thigh.	Knee jerk.
Fourth lumbar.	Internal cutaneous. Long saphenous. Obturator.	Adductors of thigh. Abductors of thigh. Flexors of knee. Peroneus longus. Tibialis anticus.	Inner side of thigh, leg and foot.	Gluteal (4th and 5th lumbar).

SEGMENT OF CORD.	NERVES.	MUSCLES.	SENSORY AREAS.	REFLEXES.
Fifth lumbar.	External popliteal. External saphenous. Musculo-cutaneous. Plantar.	Outward rotators of thigh. Flexors of knee. Flexors of ankle. Extensors of toes. Peronei.	Outer and back side of leg and foot. Sole of foot.	Ankle clonus.
First and second sacral.	Same as fifth lumbar.	Flexors and extensors of ankle. Long flexor of toes. Small foot muscles.	Same as fifth lumbar.	Plantar (5th lumbar to 2d sacral).
Third, fourth and fifth sacral.	Small sciatic, pudic. Inferior hæmorrhoidal. Inferior pudendal.	Perineal. Muscles of bladder, rectum and external genitals.	Back of thigh, anus, perineum, genital organs.	Vesical and anal centres.
Fifth sacral and coccygeal.	Coccygeal.	Coccygeus.	Skin about anus and coccyx.	Anal.

It must be borne in mind when applying this table to the practice of spinal surgery, that the several segments mentioned are not by any means exactly opposite the vertebræ bearing the same designation. This is well illustrated by the accompanying figure from Gowers' Diseases of the Spinal Cord (Fig. 27, p. 495). Referring to it we see for example that the first dorsal segment is opposite the body and spinous process of the seventh cervical vertebra. Again, we find the fifth dorsal segment opposite the body of the fourth dorsal vertebra, and the apex of the spinous process of the third dorsal. More extended remarks are unnecessary on this subject, as the tables taken in conjunction with the diagram speak for themselves.

Before proceeding with the consideration of the diagnosis of the particular systems of fibres involved, a short review of the anatomy and physiology of the spinal cord may be presented advantageously. The cord may be considered as consisting of gray and white matter, the former constituting the central mass, the anterior and posterior cornua and the gray commissure. The white matter consists of conducting fibres which connect the gray matter of the different segments with the brain above, and the nerves peripherally, and the different segments with each other. The most important portions of this structure are the following:

The direct and crossed pyramidal tracts carry motor impulses downwards from the brain, and exert an inhibitory effect on the motor cells of the anterior cornua. When, therefore, they are diseased, motor paralysis ensues, and this is associated with excessive reflex action in all spinal segments below the lesion, and spasmodic conditions in the paralyzed parts develop.

The columns of Goll "conduct special sensations from the muscles, articulations, and tendinous sheaths *via* the root on the same side. When diseased there is a loss of the sense of position of the limbs, of the power of estimating weights, and of co-ordination of muscular effort (ataxia). The fibres cross over in the medulla."

"The columns of Burdach conduct to a certain extent tactile sensations coming in from the opposite posterior root. They also contain many associative fibres, connecting different levels. Pain-sense fibres and excitoreflex from the posterior roots run through these columns to reach the commissure or the anterior horns; other fibres run through it to Clark's column and to the column of Goll. Hence it is a pathway for all kinds of afferent impressions. When diseased there may be pain, anæsthesia, ataxia, and loss of reflexes. The fibres cross over at once to the opposite side."

"The antero-lateral ascending tract conducts sensation of pain and temperature coming in from the opposite side, through the anterior and posterior commissure."

"The gray matter contains chiefly cell groups which act as centres and distributors of nerve impulses. In the anterior horns the cells have a motor and trophic function. The larger cells are at the outer parts of the horn and send fibres to the larger skeletal muscles. The more central cells are con-

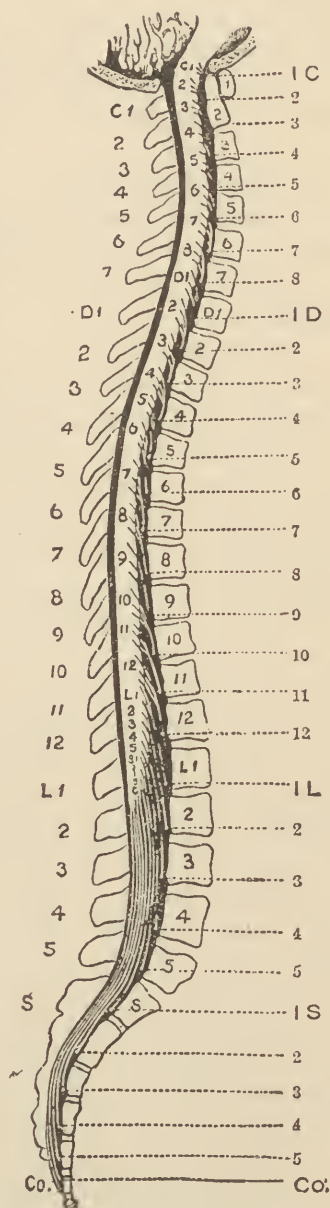


FIG. 27.

nected with small muscles and those having more delicate functions and adjustments. In the still more central and intermediate parts, also, are separate trophic cells for the muscles, bones and joints, and cell groups which preside over vaso-motor and secretory functions."



"The cells of the posterior horns are sensory in function and are connected with the tactile, pain, temperature, and reflex fibres of the posterior roots." (Dana.)

The trophic centres for the joints, bones, and skin apparently lie in the posterior horns. Their fibres pass out by the posterior roots.

Paralysis then may proceed from disease of either the motor tracts (the pyramidal tracts), the anterior cornua, or the anterior nerve-roots. In the former there is paralysis associated with a spastic condition, and exaggerated reflexes.

When the anterior cornua are diseased, the paralysis is associated with atrophy of the affected muscles.

A lesion of the pyramidal tracts causes paralysis of all parts below that lesion.

When, however, the anterior cornua only are affected, the paralysis is limited to the muscles supplied by that particular segment or segments to which the disease is limited.

The association of paralysis with rigidity must not be confounded with paralysis with contracture. In the latter condition, there exists from loss of power of certain muscles, an overaction of their antagonists, which rapidly adapt themselves to their contracted condition, undergo alterations of structure, and become contracted. An example of it is found in the deformities of acute poliomyelitis anterior.

Contracture may also occur from long-continued spasm arising from motor irritation.

Pain in conjunction with spinal affections is very often misinterpreted. Spinal pain is rarely significant of disease of the cord itself. It more frequently is due to functional disturbances, as neurasthenia and hysteria. When actual local disease is present, it is nearly always limited to the bones or the membranes.

Peripheral pains, however, have an entirely different significance. They may be due to irritation of the posterior nerve-roots, to irritation of the sensory conducting tracts in the cord, or to degenerative changes in the nerve fibres.

**Paralysis of the Sphincters.**—The centres for the bladder and rectum are found deeply situated in the gray matter of the cord, near the bases of the posterior horns. They are found in the fourth and fifth sacral segments. Destructive lesions in this locality produce paralytic incontinence of urine and fæces.

**Prognosis.**—The prospects of recovery in the majority of cases of organic nervous diseases are bad; bad because the structures diseased are so delicate that very slight pathological changes only are necessary for their permanent derangement; and bad because of the paucity of our therapeutic resources. Even functional nervous disturbances do not present as favorable a prognosis as functional diseases of other portions

of the body, probably because of the difficulty involved in determining and removing their causes.

**Treatment.**—The treatment of nervous diseases consists in one long-continued attention to detail. Every hygienic, mechanical, and medicinal resource must be employed perseveringly and persistently. It is with the greatest difficulty that the patient can be induced to persevere sufficiently or to carry out directions with sufficient exactitude to secure proper results.

In all cases, it should be the physician's aim to determine exactly what he can do and then do it. If he places before himself an impossible ideal—the rapid and complete cure of the patient—he is practically aiming at a target far beyond the range of his weapons. To recognize that he can but relieve suffering, improve the patient's general condition, or stay the course of the disease, will oftentimes yield good results.

Useless if not actually pernicious drugging is altogether too frequently resorted to because of the braggadocia of senseless empirics, therapeutic enthusiasts, and enterprising pharmacists. Of all such beware. Let one's knowledge of neurology be based on a good practical understanding of anatomy, physiology, pathology, hygiene, general therapeutics, and materia medica, and the poor nervous invalid will have his burdens sensibly lightened. We should not add to the sufferings of the patient by the *indiscriminate* use of hypnotics, analgesics, etc. (all of them boons properly given), lest we have to deal with pathological changes *plus* drug habits, a state of affairs that is well-nigh irremediable.

**Electro-Therapeutics in Relation to Nervous Diseases.**—Concerning the value of electricity as a remedy in the treatment of numerous functional and organic nervous diseases, there can be no question. Still it is a remedy that has been greatly overestimated, because of the wild claims made for it by men not versed in the slightest degree in scientific medicine. Oblivious of every pathological detail and living in complete ignorance of diagnostic neurology, they have made claims of curing the incurable, and of effecting an almost instantaneous recovery of cases tending spontaneously to that fortunate termination. Thus have physicians grown to neglect a valuable remedy. They either do not use it themselves, or they relegate its application to nurses, masseurs, the laity, or charlatans. If good results are to be obtained from electricity, it must be administered by an educated physician, thoroughly versed in the essentials of electro-physics, and by him personally.

At the present day three forms of batteries are used in medicine, the static, the faradic, and the galvanic. The first named was used considerably years ago, but fell into disuse. With improvements, in apparatus, it was resuscitated several years ago, and has now become very popular with a number of electro-therapeutists. The great cost of a proper apparatus will prevent it from coming into general use, at least for the

present, especially as it is claimed by many physicians that it can do nothing that cannot be done equally well by the galvanic battery. I myself have had no experience in its use.

The practical application of electricity in medicine involves a thorough understanding of Ohm's law, without which, one might as well prescribe medicine in ignorance of *materia medica*, or operate without a knowledge of the principles of surgery. This law is: "The quantity of electricity flowing through a circuit varies directly as the electro-motive force, and inversely as the resistance." The electro-motive force is modified in a battery by the nature of the elements used in the construction of the cells, and the strength, freshness, and consequently the activity of the liquid used to act on the positive plate. The resistance will vary according to the size and nature of the conductors. The larger the transverse surface of the conductor, the less will be its resistance. Certain metals conduct better than others; copper is practically the best in this respect, and hence it is used for making the connections in all batteries. Resistance is modified by the size of the electrode, the character of its covering, and the nature of the fluid with which it is moistened. Large electrodes conduct better than do small ones; well moistened, better than those drier; and those wet with warm or salt water, better than those saturated with plain cold water. The resistance of the human body also varies. Thus if a great portion is included between the electrodes, the resistance will be greater than if the current was made to traverse but a small distance; the skin offers greater resistance than do the subjacent tissues; and different persons offer different degrees of resistance; and the body resistance of the same person will vary from day to day. All of these statements are of importance to fix on the practitioner's mind the fact that the dose of electricity cannot be regulated by the number of cells used in the circuit or by the character of the battery. It is therefore necessary to use a milliamperemeter to determine the strength of current in use. This instrument is only adapted to the galvanic battery.

To determine the strength of the faradic battery we are at present without any suitable instrument for physicians' use. To designate dosage, we are, therefore, obliged to rely on a statement of the distance by which the coils overlap, or by the extent to which the regulating tube is drawn out.

Coming now to what will be regarded by many as the most practical portion of the subject, the first question for consideration is, which battery shall be used in any individual case? In the treatment of paralytic affections, that current should be used which will the most readily produce muscular contractions. In cases, therefore, presenting the reaction of degeneration, as paralysis dependent upon disease of a peripheral nerve or a nuclear lesion, galvanism is preferable to faradism. In hys-



terical paralyses, faradism will, as a rule, accomplish the best results. In the treatment of paralyses, the object of the peripheral applications is to stimulate local nutrition. This may best be accomplished, if the negative pole is applied to the motor points of the muscle or nerve to be acted upon, while the positive pole is applied over a higher portion of the tributary nerve, or to the cerebral or spinal centre in which that nerve finds its origin. In the case of paralysis of the legs from acute poliomyelitis, the negative electrode will be applied to the leg muscles in turn, while the positive will be placed over the lumbar enlargement of the cord. The current must be interrupted in order to secure muscular contractions. The strength employed should be sufficient to produce contractions, and no stronger. If the treatment is made too energetic, it will only result in tiring the disabled muscles.

In faradic stimulation of muscles, both electrodes may be placed over the muscle treated; in fact, they will produce less discomfort if thus used.

For central applications, galvanism is the most serviceable. Experiments have been performed to show that a current of electricity applied to the head cannot possibly influence the brain. However that may be, clinical evidence supports the value of electricity in cerebral troubles. Here, the object is to aid local nutrition. The current should be a perfectly steady one, and mild in strength. To the head, it is ordinarily not wise to exceed three milliamperes. Spinal applications may be as high as ten milliamperes with advantage.

In the treatment of neuralgias and painful affections generally, galvanism is preferable. Many have recommended faradism in these cases, but I have not been convinced of its value by their reports, and consequently have not used it. The object here is to secure the sedative influence of the current. The positive pole being the more sedative in its action is applied over the sensitive points, while the negative electrode is placed over the central portion of the affected nerve. The current must be absolutely free from interruptions.

The strength of current used in neuralgic affections is not an easy one to determine *a priori*. One must be governed entirely by results. Some cases improve on very mild ones; others only on applications of almost incredible amount. The same statement is true of the length and frequency of the sittings. As a rule, short and frequent sittings are better than long and infrequent ones. Some cases, notably those of sciatica, fail of benefit, because the current employed is altogether too weak to effect a result. Any current which produces unpleasant after-effects must be regarded as an overdose, and avoided in subsequent applications.

The size of the electrodes employed must be governed by our indications. In any given case, it must be borne in mind that the actual

quantity of current in any portion of the circuit is the same. The current must, therefore, be more concentrated at those points where the transverse section of the conductor is the least. Large electrodes, therefore, diffuse the current over a larger space, and small ones concentrate it. And again, large electrodes enable us to use a greater quantity of current with less local discomfort than do small ones. When, therefore, we wish to localize the current to single muscles or nerves, we make the active electrode a small one, the smallness being governed entirely by the indications for concentration. In cases like sciatica, where the parts to be affected lie deeply, and where considerable strength of current and diffusion are necessary to produce a beneficial result, we use very large electrodes.

Electrodes should be covered with some substance capable of absorbing water. Sponge, flannel, chamois and absorbent cotton are used. Of these, I prefer the latter, as the most cleanly and the cheapest. A fresh covering can be used for each patient. I prefer warm water to moisten them.

In most cases it is advisable to make use of both central and peripheral applications, according to indications.

In the treatment of anæsthesias the faradic brush is the most efficient agent. The stimulation produced by it exerts beneficial effects on central nutrition, examples of which will be found in the results obtained by the treatment of locomotor ataxia by this means. The wire-brush electrode should be attached to the negative pole of the faradic battery, and a current of sufficient strength to stop short of being painful, applied. The brush is passed over the anæsthetic areas, the sittings ranging in duration from five to fifteen minutes.

In cases of neurasthenia and hysteria the forms of application devised by Beard and Rockwell, and called by them central galvanization and general faradization, are exceedingly useful.

The object of *general faradization* is to bring every portion of the body in turn under the influence of the faradic current. The procedure is practised as follows: The negative pole is attached to a large copper-plate electrode, covered with a flannel moistened in warm water. On this are placed the patient's feet. The positive electrode is attached to a large sponge electrode. The seance is begun by the application of a weak current to the head. This is done through the medium of the operator's body. He takes the positive electrode in one hand, while he applies the other to the patient's forehead. He is thus enabled to judge by his own sensations of the strength of current passing. After one minute he changes the position of the hand used as an electrode to the vertex. He next places the positive pole over the back of the neck, where it is retained for four minutes. Next it is passed up and down the spine for three minutes. Then it is placed over the epigastrium for

three minutes more. Finally the seance closes by brushing the limbs with the electrode for two more minutes. The treatments should be repeated every other day. The results obtained are increased vigor, improved sleep and increased appetite.

*Central galvanization* consists in placing the negative electrode at the epigastrium, while the positive pole is applied to certain parts of the head (chiefly the vertex), to the sympathetic and pneumogastric in the neck, and down the full length of the spine from the first to the last vertebra. It is claimed to be useful in sleeplessness, neurasthenia, dyspepsia, etc.

*Galvanization of the cervical sympathetic* is a method of application designed to act on the circulation and nutrition of the brain. De Watteville and others have attempted to show the inutility of the procedure, notwithstanding which it is extensively used. Erb and Moritz Meyer are both ardent advocates of it. A medium-sized electrode is placed over the angle of the jaw, "with its surface directed backwards and upwards towards the vertebral column. The other pole should be larger and applied to the opposite side of the back of the neck, on a level with the fifth, sixth, or seventh cervical vertebra. The cathode is usually placed in front, but not always; the current should be two to five milliamperes, and the duration one to three minutes, the application stable. In certain cases, both sides may be treated successively."

In spasmodic affections, the indications are to secure the sedative influence of the current. Galvanism is here again preferable, although a number of electro-therapeutists announce a strong faith in faradism. The positive pole should be placed over the spasmodically acting nerve, while the negative is applied over its central origin. The current should be a continuous one. The results from the electrical treatment of spasmodic affections have not been satisfactory, if we exclude cases treated constitutionally by general faradization or central galvanization, in which the tonic effect alone of electricity is sought.

In some cases of spasmodic affections, central applications only are useful, *e. g.*, in chorea and epilepsy. Many claims have been made, concerning the value of electricity in the late rigidities of hemiplegia. It has been recommended that galvanism be applied to the contracted muscles, while faradism is used on their opponents. My results from this mode of treatment have been very unsatisfactory. In the various forms of tic, both galvanism and faradism have been recommended, but the majority of neurologists have obtained negative results in very many cases. My own experience has been equally disappointing.

Of late years, Corning and Peterson, of New York, have made some efforts looking to the practical application of the cataphoric action of the galvanic current. If, for example, the positive electrode of a galvanic battery be thoroughly moistened with a solution of cocaine, and the



electrodes applied to the body, the surface covered by the positive will be found in a few minutes to have become anæsthetized from the drug used. In the same way, iodine, on the positive electrode, has been used in the treatment of parenchymatous goitre. It has been proposed to extend this method of drug administration to the local medication of diseased nerves. The subject is still in its infancy, and is in need of further investigation.

Very often, prompt results are obtainable in hysterical and neurasthenic cases from purely psychic influences. The patient expects the battery to do her good, and it does. While this is a strictly legitimate use of electricity, the part played by mental suggestion must be recognized, and the curative effect credited to it, and not to the battery.

**Hydro-Therapeutics of Nervous Diseases.**—Water taken internally, or used externally as douches, baths, and packs, is a most valuable adjuvant in the treatment of nervous as of other diseases. Successful results, however, are only possible when careful attention is paid to technical details. Considering the external use of water, let me say at the outset that by means of it one can obtain either stimulation or sedative effects at will. These results are as a rule secured by reflex action through the stimulation of the skin, or influence over the cerebral and spinal circulation through the vaso-motor nerves. In a general way it may be said that cold baths exercise a stimulating, invigorating effect; warm baths, especially if prolonged, are relaxing, fatiguing, and soporific. They soothe nerve-irritability.

Proceeding now to the several methods of administering baths, etc., I will speak first of the "HALF BATH." The bathtub is filled with water at a temperature from 50° to 75° F. to a depth of about eight or ten inches. The temperature of the water must be varied according to the demands of the case. The patient seats himself in the tub, while an attendant dashes the water against him, and rubs him vigorously. Cold water is added to the bath from time to time, until the patient's teeth chatter. This method of bathing gives the stimulating effects of the cold water, the mechanical effect of the water thrown against the patient, and the beneficial influence of the frictions.

**THE COLD DOUCHE.** The value of the cold douche depends upon the application of the cold water under sufficient pressure to obtain the mechanical effects of the strong impact. A much colder temperature can be tolerated by this method of bathing than of any other. Its effects on the system are powerfully tonic. The douche at a lower temperature should never be prolonged to any greater time than one minute. The colder the water, and the shorter its application, the more complete the reaction. The effect of the douche is to deepen respiration, improve circulation, and increase secretion. It aids also in the absorption of pathological products. No remedy is a greater invigorant. It has been

shown by actual experimentation, that the cold douche increases muscular power threefold. The Scotch douche consists of the alternate douching of the body with hot and cold water. It is applicable more particularly to the absorption of pathological products, hence to chronic joint troubles. The douche is indicated, says Peterson, "in lethargic and hysterical forms of insanity, where there is sluggishness of the intellect, apathy, stupor, catalepsy, etc., in melancholic cases, and in all cases where there is anæmia, chlorosis, or gastric disorders."

THE COLD SPINAL DOUCHE is a measure that I have used with a great deal of satisfaction in neurasthenic states, especially those arising from sexual excesses. Immediately on rising in the morning, the patient is directed to go to the bathroom, to run a few inches of warm water into the bathtub, and to place a board across the sides of the tub. He now takes a position on the seat thus improvised, with his feet in the water. He then proceeds to douche the spine with water from the cold water spigot. The operation should not continue more than from thirty seconds to one minute.

THE WET PACK is practised as follows: A large woolen blanket is spread out upon the bed. Over this is placed a sheet wrung out in water at a temperature of from 50° to 70°. The patient then lies upon this, while the attendant proceeds to wrap the sheet about him, rubbing it down with his hands, so that it comes in thorough contact with the patient's body. The arms should be held somewhat from the side, so that the sheet may be inserted in the space, and keep cutaneous surfaces separated. Next, the patient is enveloped in the blanket, which should be so wrapped about him as to exclude the atmospheric air most thoroughly. The duration of the pack should be from one-half hour to an hour. The wet pack should be followed by a half-bath, or a cold ablution to restore tone to the relaxed vessels. The hot wet pack is a powerful hypnotic agent, and may be prolonged to two hours, or if the patient falls asleep in it, until he awakens.

THE PROLONGED WARM BATH consists in the prolonged immersion of the patient in water at a temperature of 90° F. Its successful practice requires careful attention to the warm water supply and escape pipes. "The calming effect of the bath has been utilized by Reiss in the treatment of over 1,000 cases of various types of brain and spinal diseases. There is no thermic effect upon the cutaneous nerves, no change of temperature, blood-pressure, cardiac action, or respiration, only a calming effect, due probably to the removal or diminution of the usual cutaneous irritations, which ordinarily are conveyed to the internal organs, especially to the nervous system, giving rise to a regulation and quieting, chiefly of the central nervous system. Cases of serious disturbance of the latter thus become amenable to treatment. Reiss has treated paraplegia of the lower extremities, paralysis of the bladder and intestines,

etc., occurring in tabes, myelitis, and similar diseases, which usually demand the use of the bath for the greater part of the day, on account of the bedsores. The latter usually healed very rapidly, if not too far advanced. But in addition, many symptoms, such as local spinal pains, eccentric pains of the extremities, distressing contractures, reflex convulsive movements, were alleviated. In similar manner, these permanent baths acted in cerebral troubles, apoplexy with chronic meningitis, hemiplegias, and with unilateral contractures also, and upon general hyper- or anæsthesias, cerebral excitement and delirium, in the most favorable manner. The calmative influence produced by neutral baths upon the condition of excitation of the brain is the chief basis of the therapeutic effect in these nervous diseases. The regulation of the peripheral irritation alone seems to suffice for the explanation of the effect; especially is the relief of insomnia a marked result of these baths." "In not a small number of cases of disease of the brain and spine, treated for weeks by permanent baths, a decided improvement of the diseased condition was noted. Motor and sensory paralysis, ataxia, and related symptoms yielded readily in these cases after failure of other treatment." "Riess gives the history of a case of compression myelitis, in which the patient spent the whole day in the bath, the night in bed, and which resulted after eight and a half months of constant treatment in complete restoration of the function of the cord!" (Baruch).

No better summary of the effects of hydro-therapeutics of nervous disease can be given than the following from Peterson:

"*Anæsthesia* (cutaneous). Short cold jet and fan douches of strong pressure to the anæsthetic areas. Temperature, 50° to 70°. Duration one minute. Daily.

"*Angio-paralytic Hyperidrosis of the Feet*. Prolonged cold foot-bath with chafing, or fan douche of cold water to the feet. Temperature, 60°. Duration, twenty minutes for bath, five minutes for douche.

"*Chorea*. Cold plunge beginning at 90°, daily reducing until 70° is reached.

"If anæmic, spinal spray, fan douche or jet, at first warm until patient becomes accustomed to them, then gradually reduced to 60° or 50°.

"*Epilepsy*. Cold shower-baths and cold sponge-baths daily are beneficial. The shower-baths should be rain-like in character—that is, not too forcible. In many cases a morning and evening bath (half-bath) proves very serviceable. When there is evidence of hyperæmia and increased blood-pressure in the head, the cold cap is useful. While these are the general indications for hydro-therapy, certain measures are often of use at the time of the seizures. During a fit of *status epilepticus*, it will be observed that there is one of two conditions present: either the face is pale and there are signs of brain anæmia, and in this case warm



wet compresses should be applied to the head and genitals, accompanied by friction of the trunk upwards, the body being placed with head low and arms uplifted; or there is turgescence of the vessels in the head, the face is red, the carotids beat strongly, and under such conditions a contrary procedure is indicated—cold compresses to the head, neck and genitals, strong wet beating of the feet with a high position of the head. Daily applications for thirty seconds.

“*Headaches, Neuralgias and Migraines.* If anæmic, heating cephalic compresses (wring out thin linen bandages in very cold water; wrap head in capelline manner, and cover with one or two layers of dry linen or flannel). Apply at bedtime. Upon removal, envelop head in dry cloth and rub it dry.

“If hyperæmic, leg bandages (a piece of towelling a yard long is dipped in cold water at one end—one-third—thoroughly wrung out and wrapped closely about each leg, so that the wet surface is next the skin and the dry portion envelops the wet two or three times. Or, wet stockings may be put on and covered with dry towels). These are applied at bedtime and retained through the night.

“*Hysteria.* For erethistic type: Wet pack, 60° to 70°, for one hour or more, followed by massage (Putnam-Jacobi); or the rain-bath at 75° to 65° for thirty-five seconds daily at twenty pounds pressure (Baruch). For depressed type: Cold effusions while standing in warm water, or hot-air bath, followed by rain-bath for thirty seconds at 85°, daily reducing until 65° is reached, this to be followed by spray douche for five seconds at 65°, or jet douche for three seconds at 65° to 55°. Reduce douche gradually to 50° or less, increasing pressure from two pounds to thirty (Baruch).

“*Hyperæsthesia* (cutaneous). Long-continued cold douches to affected area. Daily, twenty minutes, at 70° to 80°.

“*Insomnia.* Wet pack.

“*Impotence.* Brief cold sitz-baths. Daily, 56° to 64°, one to five minutes. The psychrophore, *i. e.*, application to prostate of cold by a rubber condom or bladder secured over a rectal irrigator *ou double courant*.

“*Incontinence of Urine.* In paresis of sphincter or detrusor, brief cold sitz-baths, daily, 56° to 64°, one to five minutes. Cold rain-baths (50° to 60°) and douches as general tonics. In spasmus detrusorum vesicæ, on the contrary, prolonged lukewarm sitz-baths, daily, thirty to sixty seconds, 70° to 90°.

“*Locomotor ataxia.* Prolonged warm baths, five to twenty minutes, 86° to 95°. Hot-air baths to lower extremities, followed by affusions or douches, 60° to 70°.

“*Neuralgia of all Types.* Hot-air bath to perspiration, every other day, followed by gradually lowered douches.

“*Sciatica*. Hot-air bath until patient perspires, followed by cold plunge or douche, gradually lowered to 65°.

“*Spinal Cord Affections*. In various chronic diseases of the spinal cord, the daily half-bath, 65° to 82°, six to ten minutes' duration, with affusion and chafing, will be found useful. In some cases of compression and injury to the cord, in myelitis and the like, where there is paralysis of the rectum and bladder, and formation of bedsores or trophic lesions, resort may be had with advantage to the permanent bath. A sheet fastened in a bathtub makes a hammock, in which the patient lies at first for an hour or so daily; later, all the time, except at night, when he is put to bed. The water is kept at a temperature agreeable to the patient (88°).

“*Spinal Irritation*. ‘*Douche filiforme*’ as a rubefacient and epispastic along the spinal column; or rain-baths, 65° to 85°, and douches.

“*Spermatorrhœa*. Cold sitz-baths, five to twenty minutes, 50° to 70°, daily, at bedtime; contraindicated in sexual irritability and active pollutions, where prolonged warm or hot sitz-baths, at 90° to 98°, should be used.”

**Massage** has a special value in nearly all nervous diseases. In the majority of cases, it serves a two-fold purpose: It gives the advantages of exercise, while the body is maintained at rest, and it promotes nutrition. It becomes almost a necessary adjuvant in functional nervous conditions in which overfeeding combined with rest forms the principal therapeutic means, and in organic nervous diseases generally to promote local and general nutrition. In all cases, it should be practised by a skilful masseur. Massage performed by one of the family is without value; indeed it is almost worse than useless. In many cases it is as important to consider the social as well as the scientific qualifications of the operator selected. The introduction into the sick-room of a manipulator possessed of poor intelligence and an injudicious tongue, is fraught with harm. Especially is one such harmful when claim of possession of a special magnetic touch is made. Massage must be remembered as an agent to be used on strictly physiological principles, and not as one of occult powers. In every case it should be the physician's duty to see for himself that the work is being done properly, should the masseur or masseuse as the case may be, happen to be unknown to him.

**Hypnotics** are, as a rule, very much abused remedies in the treatment of nervous states. They are probably used much more frequently than necessity demands. Sometimes, all measures fail, and then they become a positive necessity, sometimes acting as a mere makeshift, and at others doing positive good. When sleeplessness arises from pain, analgesic remedies are permissible. Of these, more will be said presently, when speaking of that class of drugs. The pure hypnotics fail

utterly to produce sleep arising from this cause. Of hypnotic remedies, I am satisfied that *sulphonal* is *facile princeps* the most efficient. I have heard physicians decry the remedy, but have found usually that it has been given by them in a very inefficient manner. The initial dose of the drug should be fifteen grains. When administered in the form of a powder, it acts very slowly and unsatisfactorily. It is my custom to dissolve the dose in two ounces of boiling water, and this solution the patient takes as soon as it is cool enough to swallow. In the majority of cases, it produces a good night's rest. Very frequently, the following day is marked by a somewhat annoying drowsiness. I do not, as a rule, think of repeating the drug again until the third night, when the medicine is administered as before. In bad cases, it may be necessary to repeat the dose every alternate night; indeed, I have often been obliged to do so. I have never thus far given thirty grains of the drug at a dose, although I would not hesitate to do so, should occasion require; but I would not in that case give it oftener than every third night. It will be found, as a rule, that the effect of the drug as a sleep-producing agent is manifest each of the nights between the doses. Sulphonal does not act very satisfactorily in sleeplessness with mania, though even here, a very good result is often obtained.

There is, it is true, another side to the action of sulphonal, but proper care in the administration of the drug, I am satisfied, will obviate any danger from that source. It should be borne in mind that before physicians use drugs of this and similar character in practice, they should acquaint themselves thoroughly with their physiological action. Then, I am satisfied, they will obtain good and avoid deleterious results.

*Hyoscine hydrobromate* is a hypnotic, useful in maniacal cases. It is best administered hypodermatically in doses ranging from the one two-hundredth to the one one-hundredth of a grain. In these doses it may be repeated at intervals not shorter than once in four hours; very often, even in violent cases, three times daily are sufficient. This drug is a peculiar one in a very important respect. The desired effect is not always obtained from the same dose. In some patients, it is necessary to give the maximum dose at the minimum interval; and, in others, the minimum dose at long intervals is required. Very often, when not properly administered, it serves to intensify instead of modify the mania. The more the condition approaches that of an active mania, the better will the drug act. Some cases resist its action, as well as the action of all other hypnotics. Then I have found that a combination of one-hundredth of a grain of hyoscine with one-quarter of a grain of morphia, hypodermically, will act surely. The sleep produced by this prescription is sometimes so profound as to be almost alarming. Coming so quickly after perhaps days of wild furor, and continuing perhaps for twelve or fourteen hours without interruption, physician and family are



alike alarmed, unless forewarned of the medicine's power. This combination I very rarely repeat; certainly, never oftener than alternate nights.

*Trional* and *tetronal* are two hypnotics which at one time it was thought would replace sulphonal. Clinical experience has not shown either of them superior to the latter remedy. Still they may be used when, as sometimes happens, sulphonal fails to act. Their dose and method of administration is the same as for sulphonal. They have a sedative as well as a hypnotic action.

*Chloralamid* is a hypnotic which may be administered when it is desirable to change drugs for a time. Its dose is fifteen grains each evening. From my use of it I have not been satisfied that it is any more efficient than others, and in many cases, does not act as thoroughly.

*Chloral*, I would not think of advising in any dose as a pure hypnotic, excepting in acute cases, in which cardiac action is good. This drug has a depressant action on the circulation, and if used persistently and in large doses may do harm. In chronic cases, it can readily lead to the formation of the chloral habit. Still in cases of weak heart I do not think, in small doses, it is as depressing as sulphonal. As a temporary palliative, it may be capable of good service.

*Morphia* and the *opium* preparations generally, are to be condemned as hypnotics.

**Rest.**—No agent in the treatment of nervous diseases possesses more value than rest; none is so universally neglected. The professional tendency is to treat cases of nervous diseases, whether functional or organic, inflammatory or degenerative, by routine exercise. Patients are ordered to keep active regardless of the apparent or concealed harm being done thereby. If they feel worse by reason of the prescription, they are told that they would have been still worse had they been kept at rest. Popular prejudice, moreover, is against rest as a remedy, for inactivity is held to be "weakening." When ordering a patient to rest for a prolonged period, one is at once treated to the objection, "Will not absolute rest in bed weaken me?" "When the time comes for me to get up will I be able to do so?" To both of these questions we can give a ready reply that rest, when indicated, is beneficial, and that it will not do any harm. It is true that a progressive degeneration may increase during the stay in bed as it did out of it; but it does so in spite of rest, and not because of it. While thus advocating rest as a great remedy, one must not go to the extreme of condemning exercise. Both agents are extremely useful in their places.

What are the indications for rest? It is my opinion that practically all organic affections are best treated by rest, either of the functionally disabled part or of the entire body. Exceptions should be made of those cases in which the disordered function has arisen from lack of use. The

tendency to treat such incurable diseases as the spinal sclerosis (locomotor ataxia, spastic paraplegia, etc.) with rest has been growing of late years. The time is as yet too early to enable one to speak positively as to the results; but thus far patients have received marked benefit. Even paralysis agitans, a disease hitherto obstinate to the best-directed therapeutic endeavors, seems to be helped by it. In some cases, the indications are for local rest mainly. An example of this is seen in sciatica, in which the rest must be enforced by the application of splints.

Pain is an absolutely certain indication of rest, and this is true whether it be of neuralgic or inflammatory origin.

In functional nervous diseases, one must be guided by indications in ordering rest or exercise. The previous habits of the patients, the influence of activity, and the state of the digestion, afford the main indications. If the patient is of a sedentary habit, if exercise benefits, or if his digestion is most excellent, exercise is most certainly called for. If, on the other hand, he has been leading an active life, he is easily fatigued, or his digestion is feeble, rest is most certainly the remedy.

To maintain a good health standard in the face of absolute rest is sometimes a difficult matter, unless a substitute for exercise be afforded. Massage here comes in as an invaluable adjuvant.

If exercise is to be ordered, its nature often requires most explicit directions. Here, every circumstance must be carefully weighed, and the results of the prescription studied carefully. Horseback or bicycle riding, billiards, swimming, walking, driving, club-swinging, boxing, or Swedish movements, each has its place. I believe that as far as possible, the exercise should be adapted to the tastes of the individual, so that while the body is active, the mind is rested. Care should be taken, too, that exercise in any one particular direction is not overdone.

**Analgesics.**—This class of remedies is far more frequently abused than are hypnotics, probably because the fanciful indications for their use are more frequent. The general rule for guidance is: "Use an analgesic when pain does not yield to other measures." As one's experience increases, he must necessarily resort to them less frequently. Many times will it be found that patients are unnecessarily dosed with this or that "pain-killer" when rest, heat, electricity, or some simple remedy, would have done the work far more efficiently.

Of the analgesics, morphia is unquestionably the most certain and most satisfactory in its action, especially when given hypodermatically in combination with a small quantity of atropia. It is very rarely, however, that it can be administered satisfactorily in neuralgic pains, because of the great liability to contract the morphia habit, if it should prove necessary to keep up the administration of the drug for any great length of time. While from one-eighth to one quarter of a grain is the dose required to begin with, it will not be many days before

one-half or even a whole grain is required to give the necessary relief. In incurable cases, in neuralgic pains dependent upon hopeless inflammatory or malignant changes, there is no remedy to replace morphia. One might as well in the beginning reconcile himself to the inevitable, and give the patient all of the little comfort that remains for him in his remaining days of life. In malignant disease of the vertebra, in sciatica dependent upon malignant disease or extensive intra-pelvic supuration, morphia becomes absolutely indispensable. While thus an ardent advocate of morphia, I must say also that I seldom use it, so far as the number of patients in whom I find it suitable is concerned. But when using it, I do so freely according to the indications given by the patient's sufferings.

Far more applicable, that is, indicated in a far greater number of cases, are the coal-tar derivatives, antipyrin, acetanilid, and phenacetin. Exalgin has also been highly praised, but I have not used it. Its dose is from one to three grains, and it is said to be very efficient as an analgesic. All of the above-mentioned remedies appear to have about the same sphere of action, although it has seemed to me that for general pains, antipyrin and acetanilid are far more efficient than phenacetin, which seems to be especially adapted to those about the head and face. While these remedies are invaluable, they must not be administered indiscriminately. The initial dose in no instance should be greater than five grains. If this produces no deleterious effects, it may be repeated. I have never given more than ten grains at one dose, and have never repeated that dose oftener than twice. It will be rare indeed that one has to give a second dose after the first one of ten grains. In every case, it is advisable to study the physiological action of these drugs; to watch the results of their administration in the sick with proper care; and to avoid giving them to patients who give evidence of cardiac weakness.

One great danger arises from the use of analgesics, hypnotics, and other palliative remedies. They so disguise or obscure symptoms that the patient is tempted to disregard nature's remedies, rest, etc., and the physician is unable to observe the progress of the disease correctly.

**Remedies in Diseases of the Nervous System.**—In presenting the general indications of a few remedies in more common use in diseases of the nervous system, one cannot refrain from making some remarks concerning the results to be expected. The physician having to do mainly with acute affections has been trained to expect quick results from treatment. If his remedies have not succeeded within twenty-four hours, he feels inclined to make a change, unless his indications are clear, and no other course is possible. With diseases of the nervous system, we are dealing with the most obstinate of all chronic diseases, obstinate in resisting both palliation and recovery. In cases of



this class we must be satisfied with doing our best, and this best is often short of our ideal. In no case do we have specifics, giving rapidly curative results, but on the contrary oftentimes we must be satisfied with stopping the course of a progressively fatal or disabling disease. The great mistake made by the majority of practitioners in this sphere of work is that of trying to effect the impossible, and thereby doing much harm.

The remedies here presented might be greatly extended, but space forbids. Other medicines will be referred to in the chapters devoted to the special diseases of the nervous system.

In very many cases, we will find it to the patient's advantage to disregard the nervous trouble altogether, and apply our therapeutic measures directly to the improvement of his constitutional condition.

HYOSCYAMUS has as important indications its mental state and convulsive phenomena. The former necessitates its use in delirium of various types in acute mania. The patient is decidedly talkative, jumping from one subject to another in the most disconnected manner. He is full of insane fears; he is suspicious of everybody and everything; fears that he will be poisoned. Often there is a highly erotic state, which leads to the most indecent behavior, even on the part of the most chaste and refined persons. In delirium tremens it is exceedingly useful when indicated by the violence of the symptoms. The use of the alkaloid, hyoscyne, has already been referred to (see page 507).

In convulsive affections, hyoscyamus is mainly useful in epilepsy, symptomatic convulsions, as those of the puerperium and infancy, and in chorea. The principal indication in each of these is the angularity of the abnormal movements.

IGNATIA is useful mainly in functional nervous states, indicated by the peculiar ignatia temperament, and etiological factors at work when it is the remedy, *i.e.*, fright, violent emotions, etc. Thus it is called for in epilepsy, eclampsia, hysteroid seizures, resulting from fright or grief. The headache is characteristically limited to a small spot and has been described as "clavis." It comes on under the influence of any emotion, and often ends with vomiting. It is relieved by copious urination.

GELSEMIUM in physiological doses produces very marked motor and sensory paralysis. These include ptosis, diplopia, difficulty of deglutition, dilatation of the pupils, amblyopia, labored respiration, and feeble action of the heart. When the doses taken are larger and the action of the drug correspondingly more profound, there are in addition, staggering gait, loss of muscular power all over the body, and diminution of sensation. The speech becomes impossible owing to paralysis of the muscles involved in that act, the heart-action more feeble and intermittent. The general paralytic effect of the drug, so far as its action outside of the distribution of the cranial nerve is concerned, shows that the drug acts on the motor and sensory portions of the spinal cord.

These effects of gelsemium suggest its use in quite a variety of paralytic affections. In ptosis (recent cases), it is our principal remedy. It is invaluable in diplopia following diphtheria, in fact in post-diphtheritic paralysis generally. Owing to the staggering gait which it is capable of producing, it has been recommended in locomotor ataxia, but I fail to see any reason why it should prove useful in this disease.

Gelsemium is a remedy for loss of power over the bladder in old people.

It may be used successfully in sexual weakness, when, notwithstanding strong erections, no emission takes place.

Among spasmodic affections, it is useful in writer's cramp, and spasm of the glottis. It has been highly recommended by allopathic authorities as a physiological remedy for spasmodic affections. In these cases, it is necessary to push the drug until it produces symptoms.

Gelsemium is well indicated in quite a variety of headaches. In hemicrania it is the remedy when the attack passes off with a flow of profuse urine, and nausea and vomiting bring relief. The pain is situated mainly in the back of the head, and is accompanied by soreness of the muscles of the neck. Often the pain extends forward through the head to the eyes. It is a useful remedy for the headaches with suffused face of the climacteric period.

In prosopalgia it is the remedy when the pain is accompanied by loss of power over facial muscles, or when the trouble has arisen from exposure to cold. The face under such circumstances is often a suffused red.

ARGENTUM NITRICUM is suited to quite a number of both functional and organic diseases. Among the former, neurasthenia with mental depression stands prominent. Physical and mental weakness are characteristic. The exciting causes of the difficulty are usually excessive indulgence in alcohol and venery. Scip, of Pittsburg, reports cases of hypochondriasis due to these causes cured by this remedy. Gastric symptoms are often present, and by many are regarded as necessary symptoms to the successful application of the drug. These consist of flatulent distension of the stomach, especially aggravated after eating; gastralgia, with flatulent distension, which interferes with the heart's action. In some of these cases, it may be necessary to use the medicine in large doses, in order to secure its local effect on the stomach. It is very often useful in pure brain-fag. Mental anxiety and profound melancholy are often present. Among organic diseases, those characterized pathologically by sclerosis come especially within the province of nitrate of silver as a homœopathic remedy. In locomotor ataxia, it is unquestionably a most useful remedy, and is thoroughly indicated symptomatically. It is less useful in other forms of sclerosis, still it may be prescribed with good effect in disseminated sclerosis of the brain or cord,

or both. In epilepsy, it has long enjoyed a good reputation, in both schools of medicine. The special indications for its use, as given in our text-books, do not strike me as at all practical. It seems best adapted to cases accompanied by the gastric symptoms above described. In the chronic stages of post-diphtheritic paralysis, it has proven useful, being indicated especially after gelsemium has accomplished all of which it is capable. It may also be used in the chronic forms of neuritis, especially those of the degenerative variety.

**GOLD.** The preparations of gold used in medicine are the triturated metal, the chloride of gold or aurum muriaticum, and the double chloride of gold and sodium, or aurum muriaticum natronatum. This remedy has far more extended uses in medicine than is ordinarily believed. In depressed mental states, melancholia with suicidal tendency, it has been used successfully by both schools of medicine. Hughes believes that the melancholia is dependent upon liver or testicular disturbance. While the drug will undoubtedly prove curative in many cases, I think that that author limits the sphere of usefulness of the remedy altogether too much. It is indicated in mental depression with disease of the testicle, either atrophy or hypertrophy, diminished sexual desires, cirrhosis of the liver, etc. It may also prove curative in primary mental disorders, and in hypochondriasis associated with gastric disturbance.

It is a remedy also for syphilitic states, more particularly however for sclerotic changes, ulcerative conditions of the mucous membranes, bone lesions, etc., than for the active recent infiltrations for which iodide of potassium is a sovereign remedy. Mercurialization is an additional indication for this medicine in syphilitic cases. Allen holds that it is in syphilitic melancholia particularly that gold finds its sphere of usefulness, advocating arsenic in the non-specific cases.

In all pathological conditions characterized by an overgrowth of connective tissue, whether affecting the brain, spinal cord, kidneys, liver, heart, etc., this remedy is indicated. Hence among nervous diseases it is called for in locomotor ataxia, disseminated sclerosis, and other sclerosis. Optic neuritis and cerebral congestions are good indications for the remedy.

Among functional nervous diseases, gold is called for in hysteria, especially when associated with hyperplasia of the uterus and ovaries.

It is strongly advocated by Bartholow in nervous dyspepsia, when there are "red glazed tongue, epigastric pains increased after food, and a tendency to relaxation of the bowels; also a duodenal catarrh and biliary catarrh, and jaundice." Vertigo accompanying gastric disturbance is an additional indication.

It may be used successfully also in neurasthenic states characterized by congestive symptoms.



The most efficient dosage is the 3x of aurum mur., or from one-twentieth to one-hundredth of a grain of the double chloride of gold and sodium, repeated three or four times daily.

*ACTEA RACEMOSA*, also known as *cimicifuga racemosa*. This remedy produces restlessness and fidgetiness, and in this condition we find one of its important indications. It is useful in chorea, especially in cases occurring in conjunction with a rheumatic history. In hysteria, with general nervousness and restlessness, it is an important remedy. It is indicated in delirium of various origins, *e. g.*, in delirium tremens, and in puerperal mania. The patient exhibits marked loquacity, with strong tendency to change from subject to subject.

Nervous troubles, associated with a rheumatic constitution, find a remedy in *actea racemosa*; *e. g.*, rheumatic torticollis, pleurodynia, and neuritis.

It is also valuable in nervous disturbances associated with disorders of the female genital organs, *e. g.*, infra-mammary neuralgia, headache and climacteric neurasthenia, the latter especially when accompanied by a sinking at the epigastrium.

The headaches are characteristically at the vertex, or the occiput. The former is usually in conjunction with uterine disorder; the latter from some reflex disturbance, or meningeal inflammation.

The neuralgias in which *actea* is useful, are quite varied, affecting any nerve or joint, but agreeing in being of reflex or rheumatic origin.

*ACONITE* and its preparations are useful in quite a variety of acute and chronic nervous diseases. Among the former class, it is indicated in those of inflammatory character in their very early stages; especially is it called for in meningeal irritation, or even inflammation arising from exposure to the sun. In these cases, the heart also bears the brunt of the trouble, and exhibits both rapidity and weakness of action.

Various motor and sensory phenomena, arising from exposure to cold winds, come within the province of *aconite*. Neuralgia in various portions of the body, and paralyses arising from inflammation of a peripheral nerve, hence especially the early stage of facial palsy, frequently find in *aconite* a curative remedy.

In chronic cases it is useful. Hysterical troubles in which symptoms are brought on by fright, and characterized by a marked sensitiveness to all external impressions, an aversion to all excitement, especially that attendant upon being in a crowd, or living on a busy thoroughfare, indicate it.

In chronic forms of neuralgia, it is as useful as in the acute. The drug seems to have an especial affinity for the fifth cranial nerves, and is curative in many painful affections affecting it, especially when associated with numbness and tingling. Even in cases dependent upon degenerative changes, it is a great therapeutic agent. No remedy acts

better than it in *tic douloureux*. Here we must often have recourse to the alkaloid, *aconitia*. In using this particular remedy, we must make certain that a reliable preparation is at our disposal, that of Duquesnel being universally acknowledged as superior to all others. The power of the remedy must be borne in mind. One should never begin with an initial dose of more than one two-hundredth of a grain twice daily. Then, the frequency of the administration may be increased each day, until physiological effects ensue, when the remedy may be continued at somewhat longer intervals. In *gouty* cases, the nitrate of *aconitia*, in doses ranging from one three-hundredth to one-thousandth of a grain, three or four times daily, may be prescribed with great benefit.

The anæsthesias, which frequently precede apoplectic seizures, afford an indication for *aconite*, by which the impending attack may often be averted by reason of the remedy's great control over the circulation. In apoplexy itself, it is useful when the circulatory symptoms are appropriate.

The spasmodic symptoms, calling for *aconite*, consist of local spasms and rigidities, and occasionally tetanus.

ALUMINA enjoys a favorable reputation in diseases of the spinal cord and especially in *tabes dorsalis*. Paralytic phenomena are marked in the symptomatology of the drug. The patient complains of paralytic weakness of the lower extremities. The legs feel heavy; or, instead of this paralytic condition, there may be inco-ordination of gait so that the patient is unable to walk in the dark without staggering. Other abnormal motor phenomena observed under alumina are tremor of the limbs, jerking and twitching of the limbs, and involuntary movements of single parts. Abnormalities in sensation may also be noted. The extremities go to sleep readily. Thus the nates go to sleep when sitting and a similar sensation may be noted in the arms. Sensation in the soles of the feet is very much blunted, giving to the patient the sensation that he is walking on cushions, or that his feet are padded. The heels become numb while walking, a sense of tightness in the arms, as if from cold, may be complained of. Pains of diverse characters may appear in the back. In one case it is as if a red-hot iron were being thrust into the spine, while in others it is as if the back had been beaten, and in still others it is of a gnawing character. The paralytic action of alumina extends even to the rectum and the genito-urinary organs. There is marked inactivity of the rectum, even a soft stool requiring inordinate effort for its expulsion. In other cases it is the function of sensation in the rectum that is impaired, as shown by the lack of desire or inability to move the bowels until there is a large accumulation of *fæces* in the rectum. Symptoms referred to the eyes, as ptosis and diplopia, may appear.

LATHYRUS has a remarkable influence over the spinal cord, notwithstanding which it has been but little used in its diseases. The

symptoms arising from poisoning with this substance will be found fully detailed in the article on lathyrism. It is sufficient to say at this time that this has led to the use of the drug in diseases of the spinal cord characterized by rigidity and excessive tendon jerks. Success has not been flattering, but then these cases are of such a nature that no treatment is likely to give satisfactory results.

**OPIUM.** The use of opium and its preparations for the relief of pain has already been commented upon. The drug may be used homœopathically for but a few nervous conditions. In the various forms of coma it is indicated, but good results cannot be expected, owing to the very serious nature of the majority, and the necessarily fatal character of the remainder of the cases in which this symptom is present. It is used by both schools of medicine in the various manifestations of uræmia. In nervous disturbances from fright (convulsions) it has been successfully used, especially in children. It is applicable to the nervous disturbances of drunkards, especially apoplexy. Of the local paralyses calling for it, paralysis of the tongue and pharynx have been especially mentioned.

The use of the remedy in mental diseases has been altogether too much neglected. It is one of the very best medicines for the cure of depressed mental states. It should here be given in the form of tincture in doses of from five to ten drops three times daily, or as morphia, in doses ranging from the sixteenth to the thirty-second of a grain at like intervals.

**GLONIN.** The therapeutic virtues of glonoin as a "nervous" remedy find reason in its action on the circulation, especially on that of the brain. It produces a high grade of cerebral congestion, with intense headaches, violent throbbing of the superficial bloodvessels, etc. It is indicated in epilepsy, neuralgia, and acute mania with violent rush of blood to the head. It is an invaluable remedy in the bad effects of exposure to the heat of the sun. It has a strong action on the mind, producing confusion of thought and loss of the sense of location, and has been employed in insanity with hot head, throbbing vessels, staring eyes and rapid pulse, especially when the same has been caused by exposure to the sun. It has been used successfully in meningitis from the above-named cause or resulting from violent emotions. It is our best remedy in cerebral vomiting. It also occupies a very useful place in the treatment of chronic renal affections and arterio-sclerosis, and exerts a wonderful action in relieving the nervous disturbances arising in conjunction with these troubles. For the headaches, convulsions and congestions, it is best used in the third centesimal dilution. In arterio-sclerosis and chronic interstitial nephritis it acts best when given in doses of one drop of the first centesimal solution three times daily. It may be advisable later to increase the dose by giving it more frequently. Some few patients cannot tolerate this initial dose, and with them it may be wise to reduce it to one-half drop to begin with.



ASAFCETIDA finds its curative sphere in functional nervous diseases, as hysteria and hypochondriasis. In its action on the digestive tract it excites a reverse peristalsis, hence it produces the globus hystericus, a bursting feeling upwards in the abdomen, etc. In hysterical conditions with tympanitic distension of the abdomen, and thoracic oppression and hypochondriasis arising therefrom, it is very efficient, as it is also in gastralgia with the same concomitants. It may also be used successfully in hysterical cough and hysterical convulsions. Especially is it applicable in cases associated with menstrual suppression. The dose must be varied according to the case. In some cases but one or two drops of the tincture are sufficient; in others it may be necessary to give thirty or more. The offensive taste of the drug need not be disguised, as in hysterical patients it is probably of some therapeutic advantage.

BORAX has not an extended use in diseases of the nervous system. It is indicated symptomatically in a number of conditions in which anxiety or nervousness with dread of a downward motion is present. Of late its use in epilepsy has been revived. It is then given in doses ranging from fifteen to thirty grains three times daily. The initial dose should, however, never be larger than fifteen grains. In other diseases than epilepsy it may be given in doses ranging from one grain of the crude drug to the third decimal trituration.

BELLADONNA. The essential character of the conditions in which belladonna is available is acuteness, either as to the course of the disease, or of the individual symptoms for the relief of which it is administered. Pathologically it is indicated in inflammatory troubles especially when localized, hence in meningitis, myelitis, cerebritis, neuritis, etc. The more violent the onset, the more likely is belladonna to be the remedy.

The head symptoms exhibit a high degree of congestion, as inferred from the throbbing and distended bloodvessels and the red face and hot head. The headaches are terrific in their intensity, the head feeling as if it would burst. They are aggravated by very slight external causes, as noise, light, etc. They are particularly violent at the back of the head or in the forehead. At times they are accompanied by temporary blindness.

In the early stages of apoplexy it is only indicated when the stupor is associated with evidences of cerebral irritation. In chorea, there is the same cerebral irritability, starting from sleep, or maniacal symptoms. These congestive symptoms serve to indicate the remedy in many cases of convulsions, both of reflex and organic origin.

The belladonna neuralgias are characterized by their suddenness of onset and departure, hence it is indicated especially in the lightning pains of ataxia.

The belladonna patient is of a plethoric build; all of his ailments are of an active type.

**ARSENICUM.** Arsenicum is used largely in the treatment of nervous diseases by the old as well as by the new school. A study of its uses would seem to indicate that it is a remedy concerning the virtues of which all can unite. Taking first the effects of the drug on the system generally, we find it producing a twofold condition, one of profound prostration or exhaustion, and the other of irritability and restlessness. It is this restlessness that indicates the drug in mental disorders, for it will be found useful in insanity with mental distress, great restlessness, constantly changing from place to place, melancholia and suicidal tendency. Sometimes the distress is characterized by extreme anxiety or fear.

Arsenic produces in both acute and chronic poisoning paralysis, which is now generally admitted to be due to multiple peripheral neuritis. Some cases, however, have been shown to be dependent upon changes in the spinal cord. Both motion and sensation are involved; the loss of power begins in the hands and feet and extends upwards to the trunk. It may be used successfully in the various forms of myelitis and neuritis.

In neuralgia arsenic is useful for both the pure type of that affection as well as for neuritis. The pains are accompanied by anæsthesia, here resembling aconite very closely, but differing from that remedy in that arsenic is especially adapted to chronic cases. Cases arising from malarial poisoning, or occurring periodically, with burning or lancinating pains, generally aggravated by application of cold, though sometimes temporarily relieved thereby, and accompanied by great anxiety and restlessness, find a great remedy in arsenic. Herpes zoster, which is now admitted to be a neurotic disorder, has in arsenic almost a specific remedy, especially when it is accompanied by these burning neuralgic pains.

In delirium tremens arsenic is occasionally useful, especially in patients long addicted to the abuse of alcohol. There are visions of ghosts and other fanciful figures, and general tremor; these symptoms are worse at night, particularly after midnight. The associated pathological changes in the kidneys, heart and liver are further indications for arsenic.

In chorea arsenic is almost the only remedy of the old school. It will be found especially adapted to chronic cases.

Arsenic may be given in tablets of the 2x or 3x triturations, at intervals of from four to eight hours. Malarial cases require the lower preparations. Many times it may be advisable to give it in the form of Fowler's solution, beginning with the minimum dose of three drops three times daily, and increasing the dose by one drop each day, until the physiological effects of the drug, pigmentation of the skin, gastric irritability, and œdema of the eyelids, are produced.

**CAUSTICUM,** although in the main a paralytic remedy, must also be

remembered as useful in neuralgic and convulsive affections. It is particularly adapted to local paralyses, *i. e.*, those dependent upon inflammation of peripheral nerves, hence in facial palsy. The efficiency of the remedy in rheumatic troubles, adapts it especially to cases of paralysis arising from that constitution. In laryngeal palsies, it is indicated when the difficulty succeeds a long-standing catarrh. It has been recommended in paralysis affecting the tongue and pharynx, but it is doubtful if it will accomplish any satisfactory results. Although useful in paralytic weakness of the bladder, it is doubtful if it will prove curative in incontinence of urine from spinal cord disease. Still, but little can be expected of any remedy in such a condition. Allen and Norton have praised causticum as a valuable remedy in paralyses of the eye, especially in ptosis of rheumatic origin.

The neuralgia of causticum occurs most frequently in the face, and is due to change of weather.

In apoplexy, causticum finds its sphere of usefulness after the initial symptoms have passed away, and the paralysis remains.

Among convulsive affections, it has been most highly recommended in chorea and epilepsy. In the former, especially in rheumatic cases, the choreic movements have been preceded by paralytic phenomena. The movements are worse on the right side, and affect face, arm and leg. In epilepsy, particularly in petit mal, with escape of urine during the attacks.

Farrington reports one case of Menière's complexus of symptoms cured by this remedy.

RANUNCULUS BULBOSUS is indicated mainly in neuralgic affections, notably in intercostal neuralgia and herpes zoster. It has been recommended in chronic alcoholism, especially for alcoholic epilepsy, hic-cough and delirium tremens.

BRYONIA. The special affinity of this remedy for the serous membranes and liver and stomach, and its curative action in rheumatic affections, explain its sphere in neurological therapeutics. In all inflammations of the meninges of the brain or cord, it is the remedy when the stage of effusion has been reached. Hence it is indicated later than belladonna. The pains are of a sharp lancinating character and are aggravated by any motion.

The headaches of bryonia are dependent upon gastric or hepatic disturbance, or are of catarrhal origin. Like all bryonia symptoms they are aggravated by motion, even of the eyeballs; the pain usually begins in or is limited to the occiput, and is of the same sharp stitching character. The aggravation is nearly always in the morning. It is very rarely indeed that bryonia is useful in a purely neuralgic pain.

ARNICA is found useful mainly in disturbances arising from traumatism, hence in many cases of neuritis, and in pathological conditions



of which extravasation of blood is the prominent feature. It is one of our principal remedies in the early stages of apoplexy, when the effused blood has produced marked evidences of compression. It promotes absorption of the clot.

In neuralgic and myalgic conditions, it is especially called for by the aching or sore character of the pain.

*BARYTA CARB.* is used largely in the degenerations of old age and the mal-developments of infancy. When as a result of defective mental development, the child fails to learn to talk or walk, and there are strong evidences of imbecility, when in old people the mind decays, memory fails, and physical and mental weakness sets in, it is indicated, though very little can be expected from it in the way of curative results.

It is a remedy in the apoplexies of old people and drunkards.

It is one of the few remedies of possible service in disseminated sclerosis. Here it is used by both schools of practice.

Its use may be suggested in epilepsies associated with marked mental impairment, in which the trouble began early in life, as a result of defective cerebral development.

*CICUTA VIROSA* has produced in poisoning cases, convulsions bearing a very close likeness to those of epilepsy. It should therefore prove useful in this obstinate disorder. There seems to be no distinctive symptoms to enable one to outline the special field to which the remedy is applicable. *Cicuta* also produces marked congestion at the base of the brain, with retraction of the head and rigidity of the neck. This has led to its successful application in cerebro-spinal fever.

It is a remedy of possible use in tetanus.

*CANNABIS INDICA* exerts a most remarkable action on the brain, producing abnormalities of perception of both space and time; a few feet seem like a mile, and a minute like hours. Guided by this symptom it has been used successfully in a number of cases of delirium tremens. It has also been given in delirium with great exaltation of mind from other causes, and with success. It is a very efficient remedy in migraine, and is used largely by old-school physicians as a means of lessening the frequency of recurrence of the attacks. To do this, it is necessary that it be given in physiological doses, *i.e.*, about one quarter of a grain of the extract three times daily.

*COCCULUS INDICUS* depends for its effects on a neutral principle which it contains—picrotoxin. It acts upon brain, medulla, and spinal cord. It produces a heavy intoxication, vertigo, inco-ordination, and diminished sensibility. In extreme cases, convulsions bearing a most remarkable resemblance to those of epilepsy occur, and are dependent upon the action of the drug on the medulla. A number of old-school physicians, among them Dujardin-Beaumetz and Hammond, have used *cocculus indicus* in this disease, with, they claim, considerable success. Of late,

there has grown up in Europe the custom of prescribing picrotoxin in combination with bromides in the treatment of epilepsy. The reported results to date, indicate apparently that the combination is a happy one.

*Cocculus indicus* produces most intense occipital headaches, and this has led to the successful application of the drug to cerebro-spinal fever.

This drug is adapted particularly to weak anæmic women. They often have pain in the small of the back, aggravated when sitting.

It may be beneficial in organic diseases of the spinal cord, when the lesions are in the lumbar enlargement, and there is marked weakness of the small of the back.

*Colchicum* is indicated in nervous affections only as such arise from the gouty or rheumatic constitution. The class of cases in which it will be found most frequently useful, are those of the peripheral nervous system, notably prosopalgia and neuritis. It has been highly recommended for gouty pains in the head, gouty headaches, with pains of throbbing or shooting character, worse on awaking in the morning. I have always used this remedy according to the directions given by Dr. Goodno, several years ago, employing the alkaloid colchicine in an alcoholic solution of one grain to the ounce, administering two drops three times daily or upwards, and increasing the dose until curative or physiological effects were produced.

*Conium maculatum* is useful in both organic and functional disorders of the nervous system. Among the former, it is indicated in paralyzes which start from below and progress upwards, an ascending paralysis, in other words. It is indicated in diphtheritic palsies, whether of the limbs or eye muscles. Sensation is not affected.

The functional nervous disturbances, in which it may be successfully used are neurasthenia and hypochondriasis, especially when such have arisen from sexual excesses or disturbances of the ovaries or testicles. It has also been recommended when these troubles have been caused by celibacy. The patient is mentally very much depressed, he is unable to concentrate his mind on his usual occupation. Often, though physically strong, sexual desire is deficient or entirely absent; erection is impossible.

It is adapted to the mental deterioration of old age. Vertigo is a prominent indication. This latter symptom is aggravated by any motion or even by turning over in bed. When conium is indicated, it is caused by old age, or by sexual excesses.

The dominant school has made considerable use of conium of late years as a depresso-motor, giving the remedy in doses sufficient to produce the physiological effects, *i. e.*, weakness of the lower extremities. To do this, it must be given in the form of fluid-extract. Care must be taken in this case, that the preparation is a reliable one, as fluid extracts of conium vary greatly in power.

Owing to its reputed efficacy in malignant growths, it may be employed in the treatment of cerebral tumors.

CUPRUM may be used in convulsive, paralytic, or neuralgic affections. In the former it is especially indicated when spasm of the flexor muscles predominates, and when the convulsion is attended with blueness of the surface, and the paroxysm is followed by deep sleep. It is indicated in epilepsy, reflex convulsions, and uræmia.

Local spasms also find in it a remedy, especially when involving the calves.

It is useful in chorea when that affection has been brought on by fright.

In epilepsy, it is called for when the convulsive movements are general, involvement of the flexor muscles predominates, the attack is ushered in by the epileptic cry, and is followed by a prolonged sleep. The hands are strongly clenched.

The cuprum meningitis is accompanied by a violent delirium, and is especially liable to complicate the exanthemata, as scarlatina and measles.

The paralysis of cuprum attacks the flexor muscles, and is accompanied by atrophy. It is well indicated in amyotrophic lateral sclerosis.

It may prove of possible use in paralysis of the tongue, especially when associated with general paralysis of the insane. Speech is stuttering.

In neuralgic affections copper is of some use, but here the best results will be obtained from the arsenite, which is valuable in visceral neuralgias.

DIGITALIS is indicated in nervous affections by the character of the pulse, which must be either slow, or feeble and quick. It is especially suited to meningitis and cerebro-spinal fever. The retinæ are congested; the pupils dilated; coma, great prostration with coldness; throbbing headache; delirium.

It is a most excellent remedy for seminal emissions. Here it should be given as digitaline, as directed by Bæhr, two grains of the third decimal trituration each evening.

In disturbed sleep it is oftentimes useful. The patient arouses from sleep distressed with a feeling perhaps that he is falling from a height; he feels anxious and apprehensive; breathing is deep and sighing.

The association of nervous disturbances with disease of the heart or kidneys makes a strong indication for digitalis.

PHOSPHORUS is a remedy of very extended use, being adapted to quite a variety of functional and degenerative diseases of the nervous system. Its sphere of action may be summed up as follows: Fatty degenerations, vascular changes producing organic disease, exhaustion with morbidly rapid growth, and associated with an abnormal hyperæsthesia.

In neurasthenia and its congeners, it is indicated when the condi-



tion is caused by excessive work or seminal losses, too rapid growth, acute infectious diseases, rapid child-bearing, etc. The back feels weak as if it would give out, and there are burning along the spine, sexual excitement, throbbing headache, and weakness in the epigastrium. There is a morbid impressionability so that the slightest odors, sounds, etc., aggravate symptoms, and emotional influences produce cardiac palpitation. When sexual exhaustion is present, it will be found that it is accompanied by excessive sexual desire, or has been preceded by this condition.

In hemiplegias, locomotor ataxia, and other organic diseases, it is a remedy, being adapted to the nerve lesions itself as well as to those liable to accompany it, as vascular degeneration, Bright's disease, etc.

In mental troubles it is adapted to general paralysis of the insane and to mania with erotism.

Eye symptoms, as amblyopia, optic nerve atrophy, and night blindness, are indications for phosphorus.

PHYSOSTIGMA, also known as calabar bean, has a double use in practice, being adapted to the treatment of tetanus and functional diseases of the spinal cord. In the former, it is largely used by allopathic physicians, though in physiological doses. In hysterical spinal affections, it is indicated by numbness of the feet and hands, and crampy pains, rigidity of the muscles of the back, twitching of the muscles of the extremities when dropping off to sleep, and localized sensitiveness of the back.

PLATINA is of use mainly in hysterical and ovarian diseases. It is indicated by anxiety and apprehensiveness. The digestive tract shows abnormalities in the shape of flatulence and constipation. In hysteria and melancholia associated with uterine and ovarian disorders, it is one of our main remedies, corresponding closely to the action of aurum on the male genital organs. There is almost always increased sexual excitement, amounting in some cases to nymphomania.

In paralyses, it is indicated when they are associated with localized anæsthesias.

It is useful in neuralgia, when associated with the numbness just referred to, and when the pains are of a crampy character. The pains increase and decrease gradually. It is one of our most important remedies for the nervous affections following unnatural sexual vices.

RHUS TOXICODENDRON is indicated in quite a variety of nervous conditions, most of which, however, are due to the rheumatic diathesis. In paralysis dependent upon neuritis or perineuritis, especially when following exposure to wet or cold, or overwork, it is the remedy to be first thought of. It is also frequently indicated in the paralyses following apoplectic seizures. The local palsies in which it is most frequently indicated, are ptosis, paralysis of the extrinsic muscles of the eyeballs, facial paralysis, and paralysis of the muscles of the larynx. The same indications call for its use in quite a variety of neuralgias and headaches. •

It is an important remedy in the vertigo of old people, especially when associated with a sensation as of swashing in the brain.

ZINC is adapted to quite a variety of neuralgias, spinal affections, and degenerations. Its effect on the spinal cord will be fully explained in the chapter on locomotor ataxia.

STRAMONIUM finds its therapeutic sphere in spasmodic and excited conditions, as chorea, epilepsy, delirium tremens, mania, etc. It is characterized especially in all these cases by the violence of the symptoms.

**Surgical Measures in Diseases of the Nervous System.**—Recent years have been marked by great advances in the application of surgical principles to diseases of the central and peripheral nervous system. The enthusiasm created by this new field of labor tends without doubt to the adoption of too radical measures, and the doing of considerable damage. But the same is true of all new discoveries. Brain surgery may now be said to have reached the period of its career when a wise conservatism prevails in its practice. The necessity for operative treatment in the majority of cases of fracture of the skull is now pretty generally recognized. In cases of compound depressed fracture, there seems to be no difference of opinion as to the proper course to be pursued. The same is true of simple fracture when attended by evidence of cerebral disturbance. But in the case of simple depressed fractures without marked symptoms, authorities are at variance. The opinion is, however, growing that even these cases should be operated. This radical measure has strong reason for its endorsement, as is shown by the many cases of head injury which develop epilepsy, and other serious conditions in after-years. In view of this, and especially when it is well recognized at the present time that secondary operations for the cure of traumatic epilepsy are successful only in a very small percentage of cases, it seems to me that the views of those who recommend operation in every case of depressed fracture are entitled to the greatest respect. Whenever there exists a doubt as to the proper course to be pursued, I think that the doubt should be decided in favor of at least an exploratory incision. With very marked cerebral symptoms, even trephining should be performed, of course guided by intelligent surgical and neurological indications.

In cases of injury with laceration of the brain structure, every care must be taken to prevent the formation of cicatricial tissue within the structure of that organ. Such tissue, it is well known, is very apt to cause epilepsy. Even the adhesions between the membranes following an ordinary trephining have produced epilepsy. Ragged pieces of brain must, therefore, be trimmed off, of course using proper judgment. Objections to this procedure have been made because of the danger of thereby damaging cerebral functions. This danger is more apparent than real, for where portions of the cerebral cortex have been excised

for therapeutic purposes, the loss of the corresponding function has been but temporary.

Intracranial hæmorrhage is an accident for the benefit of which much has been expected from surgery. The poor prospects of any relief thereby in the case of intra-cerebral hæmorrhage will be fully appreciated after perusal of the chapter on "apoplexy." In the case of meningeal hæmorrhage, the prospects of relief from trephining and removal of the clot are, as a rule, excellent, especially if there be no accompanying injury of brain substance. Even when the extravasation in such cases is not sufficient to cause death, operation is wise as a preventive of permanent impairment of cerebral functions; for where so much cortex is affected, as there must be in cerebral hæmorrhage, perfect restoration to health cannot be expected under a too conservative treatment.

The treatment of hydrocephalus by operation will be considered in the special article devoted to that subject.

The treatment of idiocy by craniectomy must be regarded as utterly useless. In a number of cases in which I have been associated with my friend Dr. W. B. Van Lennep, the results have not been such as to warrant further resort to such a dangerous operation.

As to the operative treatment of tumors and abscesses of the brain there can be no question. The results are in the main good, when the infancy of the subject is taken into consideration.

Spinal surgery also has a great field in the future. At this time, I shall speak only of the operative treatment of traumatic paraplegias. When a spinal cord has been damaged by fracture or dislocation of the vertebra, but little can be expected from operation after the lapse of any great interval of time. Under such circumstances, there usually ensues extensive degeneration of the delicate fibres of the various systems of the cord, and these can never be replaced, no matter how thoroughly pressure and irritation may be removed by trephining. Early operation is indicated; but then we run the risk of shock from the procedure, the shock being all the more serious by reason of its taking place so shortly after the original injury.

Nerve resection and nerve stretching have indications in the treatment of neuralgias. The latter is, as a rule, of doubtful utility, and is to be regarded as a means to be adopted in selected cases. For further information concerning both operations, the reader is referred to the chapters devoted to the consideration of the special forms of neuralgia.

Surgical operations performed for the cure of reflex conditions are, as a rule, to be condemned unless the condition of the part operated upon is such as to indicate the procedure regardless of the existence of the associated nervous condition. We hear much of this and that trouble cured rapidly by this or that operation, and yet we find that such cases but rarely occur in the experience of advanced students of medicine.



The relation of status of reflexes in the etiology and treatment of disease is best stated in the language of Mitchell: "The strange causes, the reflex visceral parentages, the lucky decayed tooth, etc., are, alas, too rare, but serve to keep us watchful and to make the text-books less dull reading by introducing the pleasant unexpectedness of romance." An experience by no means small leads me to say that the reflex cases are few in number, and that the wild resort to operations in this or that special field is productive of negative, if not indeed harmful, results.

# AFFECTIONS OF THE CRANIAL NERVES.

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## AFFECTIONS OF THE OLFACTORY NERVES.

Very little definite information concerning the disorders of the olfactory nerves and the significance of olfactory symptoms is in our possession. Impairment of the sense of smell in the direction of deficiency causes so little discomfort that the patient rarely if ever consults the physician respecting the same. While in cases in which the sense of smell is hyperacute or perverted, other symptoms of far greater importance generally overshadow it. The investigation of the olfactory function is furthermore unreliable, no satisfactory instruments or methods for this purpose having been devised. It is true that Zwaademaker has invented an olfactometer of some value. It fails, however, to establish a normal standard by which abnormalities may be gauged. It consists of two tubes, one sliding within the other. The inner tube is coated with some odoriferous substance. The nozzle is inserted into the nostril while the patient inhales. When the inner tube is withdrawn the passage way for the inhaled air is longer and fewer odoriferous particles can reach the nasal cavity. The sensibility of the function is estimated by the extent of withdrawal of the tube. The greater the distance at which the tube may be withdrawn and the odor perceived, the more acute the olfactory sense.

In testing the sense of smell, care should be taken to employ as test substances only such as are non-irritating to the nasal mucous membrane. Those best adapted for the purpose are oil of cloves, oil of bergamot, camphor and musk.

Alterations in the sense of smell may take place as follows: Loss of sense of smell or *anosmia*; hyperacusis or *hyperosmia*; perversions or *parosmia*.

(a) **Anosmia** is generally the result of disease of the nasal mucous membrane. Still it may arise from lesions in the olfactory nerve or bulb, or in the cortical olfactory sense centre, at the tip of the temporo-sphenoidal lobe. It has been known to accompany meningitis, tumor of the brain involving the olfactory bulb or nerve, caries of the bones, injuries to the head, and locomotor ataxia. In the latter disease it is probably the result of atrophy of the olfactory nerve.

(b) **Hyperosmia** occurs especially in conjunction with hysteria, and in certain hypnotic states. It is claimed that such patients are sometimes able to perceive plainly odors that to others are undiscernible.

(c) **Parosmia**.—Under this head are included hallucinations of sensations of odors. These are especially liable to be found in the insane, and may constitute the aura of some cases of epilepsy. Occurring in the latter connection and in association with organic diseases of the brain, they are of importance as localizing symptoms. The odor is nearly always of a disagreeable character.

Hyperalgesia of the olfactory sense is sometimes noted. In this condition odors normally pleasant become unbearable, if not distressing.



## LESIONS OF THE RETINA AND OPTIC NERVE.

### LESIONS OF THE RETINA.

Although it is very generally considered that diseases of the retina are of a character requiring the technical knowledge of the ophthalmologist for their clinical investigation, they are in many instances the direct results of systemic and visceral diseases, and are therefore of the highest importance to the general practitioner. Ofttimes, indeed, they sound the warning notes of an oncoming or already well-developed disease, the previous existence of which had not been suspected. The affections which especially concern us are retinitis and the various functional disturbances of the retina.

**Retinitis.**—This is not infrequently present in Bright's disease, especially in that form known as interstitial nephritis, diabetes, syphilis, and the various anæmias. Its recognition depends entirely upon ophthalmoscopic examination. Its characteristic symptom is a loss of transparency of the retina as shown by diffused haziness or circumscribed opacities and swellings. In advanced cases there are areas of exudation exhibited as white spots, which may be confluent or discrete. Changes in the bloodvessels, when present, take the form of increased tortuosity when passing over swollen or infiltrated tissues, and are obscured in the infiltrated parts. Hæmorrhages may be present and invade either the nerve-fibre layer or the deeper portion of the retina. In the former case, they exhibit frayed edges; in the latter they are more compact, rounded, and have clear-cut margins. In old cases, the hæmorrhagic spots are replaced by deposits of pigment. The disk may or may not give evidences of inflammation.

Retinitis is spoken of as retinitis albuminurica, leukæmic retinitis, and syphilitic retinitis, according to the cause producing it. It is generally conceded that retinal changes are present in about one-fifth of the cases of interstitial nephritis. Very often, indeed, its attendant disturbance of vision is the first symptom to direct the patient's attention to the fact that he is not in good health. It is characterized objectively by the grouping of white spots about the macula. Some cases present a high degree of attendant neuritis, which will, unless the physician exercise unusual care, lead to the oversight of the actual condition, and owing to the associated cerebral symptoms, lead to a mistaken diagnosis of brain tumor. Retinitis albuminurica is, in the majority of cases, symptomatic of advanced renal changes. The exception is found in those instances

in which the nephritis is but one of the conditions attendant upon widespread arterio-sclerosis.

The TREATMENT of retinitis is found in absolute rest of the eyes, and careful attention to the causative factors.

**Functional Disturbances of the Retina.**—These consist of toxic amaurosis, hysterical amblyopia, hyperæsthesia of the retina, and hemeralopia and nyctalopia. Amaurosis and amblyopia may be defined as conditions in which there is loss or diminution of vision without ophthalmoscopic changes to account for the same.

TOXIC AMAUROSIS is of not infrequent occurrence as a manifestation of uræmia. It often comes on suddenly, indeed the patient may wake finding himself blind. While frequently associated with convulsions, it may be the single manifestation of the toxæmia. As a rule it disappears when the function of the kidneys is restored.

Amaurosis may also result from lead, quinine, and salicylic acid poisoning. Arising from the first named, it is of very rare occurrence. When lead affects the vision, it usually does so by first producing optic nerve atrophy or neuritis.

The frequency of *tobacco amaurosis* has been variously estimated. Some regard it as of very rare occurrence; others believe it to be a not very uncommonly observed condition. It occurs especially in persons who have also indulged to excess in alcohol. Chewing tobacco seems to be more injurious in this direction than is smoking. It is characterized clinically by obscuration of central vision, especially for colors, notably red and green. If not properly treated, organic changes in the optic nerve may develop.

*Hysterical amblyopia* may be either complete or incomplete, temporary or permanent. It is characterized by limitation of the visual fields. It is usually associated with other hysterical symptoms, although it may occur as an isolated symptom.

RETINAL HYPERÆSTHESIA is symptomatic of hysteria, and of weak states of the system generally. Strictly speaking, the term hyperæsthesia as applied to the retina should convey the idea of increased acuity of vision, a condition that is of the rarest kind. It is really applied to a state in which light produces unpleasant effects. It is to be distinguished from photophobia accompanying affection of the outer tunics of the eyes.

REFLEX AMBYLOPIA has been observed in cases of irritation in the course of some of the cranial nerves, notably the fifth, and in diseases of the stomach.

NYCTALOPIA is a condition in which objects are seen clearly during the day, but not in the subdued light of evening.

HEMERALOPIA is the reverse of nyctalopia.

## LESIONS OF THE OPTIC NERVE.

### **Inflammation of the Optic Nerve ; Optic Neuritis ; Papillitis.**—

Optic neuritis may occur as an idiopathic affection (though very rarely), and as symptomatic of quite a variety of diseases, notably, of tumor of the brain. It is also observed in quite a variety of cerebral conditions, as meningitis, abscess, thrombosis, etc. It has been observed consecutively to certain acute febrile diseases, especially scarlatina, typhoid fever, variola, etc. It is sometimes observed as an essential symptom of syphilis. Cases have arisen from exposure to cold in persons of a rheumatic constitution. In women, it is sometimes symptomatic of anæmia and menstrual disturbances. Local causes as orbital diseases, *e. g.*, tumor, caries, periostitis, etc., explain some cases.

In tumor of the brain, it is a highly important symptom, being present in a majority of cases.

The DIAGNOSIS of optic neuritis depends exclusively upon the ophthalmoscopic examination. The disk exhibits an increased redness, and its borders are obscured. Swelling is next observed, and this increases until, finally, the form of the disk is lost entirely. Its location is recognized only by the point of convergence of the vessels. The arteries are usually not increased in size, and are always partially concealed by the swelling. The veins are distended and tortuous. Hæmorrhages are often observed.

Sight is not necessarily disturbed. Cases have been observed in which acuity of vision, the color sense, and the normal fields, have all been preserved. Usually, however, all of these are affected. The extent to which sight is disturbed, varies greatly in different cases. Very often, vision does not show marked diminution until the stage of subsidence of inflammation.

The PROGNOSIS depends entirely upon the causative factors at work. Slight cases may subside, leaving no disturbance of vision. Severe cases may go on to absolute blindness.

**Optic Nerve Atrophy.**—Optic nerve atrophy may be either primary, or secondary. As a primary affection, it may be idiopathic, or a symptom of degenerative affections, as locomotor ataxia, disseminated sclerosis, and general paralysis of the insane.

As a secondary affection, it is most frequently observed after neuritis, and in lesions of the cortex and chiasm.



## THE MOTOR NERVES OF THE EYES.

The cranial nerves through which the eyes receive their motor power are the *fourth* or *patheticus*, which supplies the superior oblique muscle; the *sixth* or *abducens*, which goes to the external rectus; and the *third*, or *motor oculi*, which supplies the remaining muscles, both external and internal. Paralysis of any one of the ocular muscles produces certain well-defined symptoms, differing in character, however, according to the special muscle affected. It will conduce to a readier understanding of what is generally regarded as a very intricate subject, I believe, if I first detail the symptoms of paralysis of the motor nerves of the eye in general, leaving to the last of this section a description of disease of special nerves and muscles.

**Etiology.**—Paralyses of the ocular nerves may be congenital or acquired. The great majority of the latter cases are syphilitic in origin, the remainder being rheumatic or secondary to some infectious disease, especially to diphtheria. Some few result from organic brain disease, as tumors of a sarcomatous or tuberculous nature. In childhood the majority of cases of ocular paralyses are of tuberculous origin. Occurring as a symptomatic condition, these palsies are observed during the course of locomotor ataxia, disseminated sclerosis, general paralysis of the insane, and some other central organic affections.

**Pathology and Morbid Anatomy.**—A complete exposition of the pathology of oculo-motor paralysis involves far more lengthy explanation than space will permit. It is only possible here to refer to the subject in the most general way. Ocular paralysis may result from focal or general lesions of almost any character. If the former, the damage may be cortical, in the nerve nuclei themselves, in the association fibres, in the fibres between the nuclei and point of emergence of the nerve from the brain, in the course of the nerves at the base of the brain or through the foramina or within the orbit. Syphilitic and tuberculous processes affecting, as they are especially apt to do, the basal meninges, are liable to interfere with the function of these nerves, all of which travel an unusually long and exposed course before leaving the cranial cavity. In some cases we find small foci of degeneration damaging the nuclei or the various systems of fibres. Still others are of very uncertain pathological nature, certain rare transient and recurring paralyses of the third pair of nerves being deserving of especial mention in this connection.

**Symptoms.**—The one cardinal symptom of paralysis of an ocular nerve is *strabismus*—a strabismus that presents certain points of distinc-

tion from the ordinary concomitant squint, so common as the result of refractive errors. The paralytic squint is marked by defective movement of the eyeball in the direction of action of the affected muscle or muscles, diplopia, false projection of the visual field, and greater secondary deviation of the sound eye. Each of these conditions requires explanation.

(a) *Defective movement in the direction of action of the paralyzed muscle or muscles.* In the great majority of cases of paralysis of eye-muscles, the deviation of the eye is patent to even the most careless observer. The general appearance of the deformity does not differ, however, from that of concomitant squint until certain tests have been applied. By holding an object before the patient, and moving the same in different directions while the patient follows it with his eyes, the activity of the muscles of these organs may be readily judged. Care must be taken, however, that the patient does not move his head, even in the slightest degree, while this observation is being made.

(b) *Diplopia.* This is always present in paralytic squint of comparatively recent onset. In old cases, the patient learns to disregard the false image and sees only the true. In many such, however, the diplopia can be restored by placing a red glass before one eye, preferably the sound one. The relative positions of the true and false objects will vary according to the extent of the lesion and the muscle involved. If the paralysis is but a very slight one, the separation of the images is correspondingly small. The false image may be above or below or to either side of the true one; or it may be inclined at angle to the true image, and still in any of the above-named relative positions.

It is customary to speak of diplopia as *simple* or *homonymous* and *crossed diplopia*. Simple or homonymous diplopia is that variety in which the right hand image is seen by the right eye, and the left hand image by the left eye. In crossed diplopia the right eye sees the left hand image, and the left eye the right hand one. Homonymous diplopia is produced by convergent squint; and crossed diplopia by divergent squint. This statement is a simple one, but can readily escape the memory. For our guidance we have a rule formulated by Gowers, and this is: "*When the axes of the eyes if prolonged, cross, then the images are not crossed; and vice versa.*"

In order to distinguish to which eye each image belongs, it is only necessary to place a red glass before one eye, which will color the corresponding image.

To determine the extent of the paralysis and its variations from day to day, several methods are at our disposal. We may measure it by means of a little instrument consisting of an arc with degrees marked on it; or by means of a perimeter determine the field over which the patient has binocular vision of the test object. Lastly we may deter-

mine the degree of the prism required to overcome the double vision. In applying the latter test, it should be remembered that the base of the prism should be placed in the direction of the weakened muscle.

A patient with diplopia assumes peculiar attitudes of the head that he may overcome the visual error, in part at least. So characteristic is this that a skilled observer can sometimes by this alone determine the trouble. The head is carried in the direction of the action of the paralyzed muscle. Thus in paralysis of the right external rectus the head is apt to be turned to the right, because in that position the diplopia is at a minimum, if not entirely obviated. Looking towards the left will intensify the symptom.

(c) *False projection of the visual field.* By this expression we mean to convey the idea that the ability of the patient to judge correctly of the position of objects is impaired. In health, we are enabled by our visual apparatus to judge precisely of the position of an object before us, and reach for it correctly. We do this by estimating the amount of muscular exertion required by the eyes in bringing their axes to bear on it. This is done unconsciously, of course. If the eyes are at rest in raised position, and an object is in front of it, we know that the object is in front of our face. If that object is moved to the right, we can judge, by following it with our eyes, of the extent of that change. If, however, the right external rectus is paralyzed and we attempt to follow the object, we require greater innervation than is normally given to that muscle; and, judging by the extra exertion thus made, we are led to locate the object much further to the right than it really is and make a reach for it accordingly. This false interpretation of position leads to a not infrequently experienced symptom in ocular palsies, namely, vertigo. To overcome this the patient is often led to close the affected eye.

(d) *The secondary deviation of the sound eye is greater than the primary deviation of the affected eye.* If, in a case of paralytic squint, the patient fixes with the sound eye, a deviation will be observed in the affected one. This is the primary deviation. If now the sound eye is shielded, and the patient made to fix with the one affected, the former deviates, and it will be observed that this deviation is greater than that observed in the affected eye. This is the secondary deviation. These phenomena are easily explained. In health, an equal amount of nerve force is sent to each eye when the subject is required to fix with either. If, in a case of ocular paralysis, the patient fixes with the well eye, only the normal amount of nerve force is expended in making the movement and goes to each organ. The diseased eye exhibits a deviation corresponding to the intensity of the paralysis. To fix with the affected eye requires an expenditure of much greater than the normal force, and this is transmitted likewise to the normal one. Consequently, acting under its influence, it exhibits far greater deviation than did its fellow.



It would seem that with a little care and experience the recognition of ocular palsies is an easy matter. This is true in the majority of cases when seen early. But circumstances will arise in which the most expert will be utterly unable to determine the precise condition present. Complicating factors creep in, and, as it were, upset every possible calculation. Several muscles may be affected to unequal degrees, in some the paralysis being complete, in others partial. This offers a difficult problem for solution. Sometimes paralysis sets in in cases in which the muscular equilibrium had previously been affected. This muscular insufficiency is reflected in the character of the diplopia. Sometimes the trouble appears in patients whose eyes have very unequal visual powers, and the new lesion affects the better eye. The patient is then obliged to use the latter for fixation, and the amblyopic eye is in a state of secondary deviation. The diagnostic danger in such a case is that of considering the sound eye the paralyzed one.

In old cases the unaffected muscles often undergo secondary contractures, producing almost any possible deviation. This impresses itself on the diplopia, the *modus operandi* of which becomes almost unfathomable.

**Paralysis of the Individual Oculo-motor Nerves.—Third Nerve.**—This nerve supplies all the muscles of the eye with the exception of the external muscle rectus and the superior oblique. All of the movements of the eye are therefore abolished, with the exception of abduction and rotation downwards and inwards. The power of accommodation is lost and the pupil is moderately dilated and does not react to light.

When the paralysis is complete, there is drooping of the upper lid (ptosis), deviation of the eyeball outwards and downwards. The diplopia is crossed. The two images are on the same plane when the object is horizontal with the eyes. When, however, the object is below, the false image appears below the true; and when above, the true image occupies the lower position. Sometimes, when all of the eye muscles are paralyzed, there is a slight protrusion of the eyeball (*exophthalmos paralyticus*).

*Paralysis of the levator of the upper lid* occurs in quite a variety of conditions. It is probably observed as a symptom of locomotor ataxia or hysteria more frequently than under other circumstances. It may also result from nuclear disease, in which case it is apt to be associated with paralysis of the superior rectus. It has been seen as a congenital condition, these cases being practically incurable. A few examples arise from cortical lesion. A transient drooping of the upper lid is observed in some delicate women, especially in the morning.

*Paralysis of the superior rectus* gives rise to a deviation of the eye downwards, with slight divergence owing to the unopposed action of the inferior oblique. The diplopia is aggravated when the patient attempts

to look upwards. The false image is seen above the true, and is tilted, in the case of the affection of the left eye, to the patient's right.

*Paralysis of the internal rectus* produces divergent strabismus. The diplopia is crossed.

In *paralysis of the inferior rectus* the affected eye deviates upwards and slightly outwards. The relative positions of the images is the exact converse of that given above for paralysis of the superior rectus.

In *paralysis of the inferior oblique* the defect in movement is upwards and inwards. It occurs very rarely as an isolated condition, and when associated with other ocular palsies is difficult of detection.

**Fourth Nerve.**—This supplies the superior oblique muscle, paralysis of which causes a loss of movement downwards and inwards. The strabismus is convergent, and is observed especially when the patient looks downwards, there then appearing a simple diplopia. This condition is very liable to be mistaken for paralysis of the superior rectus; in the latter case, however, the loss of movement is downwards and outwards, and the diplopia is of the crossed variety, and is aggravated when the eyes are directed upwards.

**Sixth Nerve.**—Paralysis of the sixth nerve causes loss of power in the external rectus, with a convergent squint and homonymous diplopia.

**Diagnosis of the Seat of the Lesion.**—Cortical lesions do not produce ocular palsies other than ptosis, and even this is rare from such a cause. When the lesion is nuclear, a number of muscles are generally affected. One external muscle after another is affected, leaving the internal muscles unaffected. Nuclear involvement of but one muscle is almost always confined to locomotor ataxia, disseminated sclerosis, etc. A fascicular paralysis, that is one dependent upon damage between the nuclei and the point of emergence from the brain, is exhibited by alternate hemiplegia. In the case of the third nerve, the oculo-motor paralysis on one side, and that of the face, arm and leg on the other; in the case of the abducens, there is an alternate paralysis also, the parts affected being the external rectus and face on one side, and the arm and leg on the other. When the nerves are involved in their course at the base of the brain, one cranial nerve is attacked after another; the invasion of the trigeminus is exhibited by neuralgia as an introductory symptom; blindness appears in one eye, without ophthalmoscopic changes. When the lesion is in the orbit, the associated symptoms must decide. Local pain, exophthalmos, optic neuritis, etc., are suggestive.

**Ophthalmoplegia.**—This is a term first employed by Jonathan Hutchinson to designate a progressive paralysis of all the eye muscles, and dependent upon nuclear degeneration, hence it has been spoken of as *nuclear ophthalmoplegia*. When the external muscles of the eye only

are affected, it is called *ophthalmoplegia externa*, and when limited to the internal muscles, *ophthalmoplegia interna*; and when all of the muscles are involved, *total ophthalmoplegia*.

Nuclear ophthalmoplegia has been observed most frequently as one of the concomitants of locomotor ataxia, general paralysis of the insane, and progressive muscular atrophy. Many cases are of syphilitic origin. It sometimes follows diphtheria. The prognosis is unfavorable.

**Prognosis.**—The prognosis of ocular paralyses must depend upon their duration and cause. Those accompanying locomotor ataxia nearly always recover, though they are liable to relapse. Nearly all the syphilitic and rheumatic cases recover under suitable treatment. Cases dependent upon degenerative changes or tumors other than gummata are of ill omen.

**Treatment.**—The treatment must be regulated by the nature of the pathological changes. Syphilitic cases must receive *energetic anti-syphilitic* treatment. (See section on syphilis of the nervous system, p. 440.) Rheumatic cases will recover under *rhhus*, *causticum*, *kalmia*, and *gelsemium*. Post-diphtheritic diplopias yield to *gelsemium* or *argentum nitricum*. Degenerative changes in the nuclei call for *argentum nitricum*, *aurum mur*, or the *double chloride of gold and sodium* in doses ranging from one-twentieth to one one-hundredth of a grain three times daily.

When the diplopia becomes so annoying as to interfere seriously with the patient's comfort or daily work, a ground glass should be worn in front of the diseased eye.

Incurable cases may be treated by surgical operation, but care must be exercised not to promise too much as a result. Even though the eye may be restored to its normal position, we must not forget that it is still guided by weakened muscles, and the new equilibrium may not be preserved in all positions. An æsthetic effect is, of course, obtained, but, excepting in the case of women, this will not be appreciated, if it is done at the expense of personal comfort. The patient may have learned to disregard the false image by reason of the long continuance of his squint. In this case, the operation often causes a return of the diplopia, if it does not effect an exact adjustment.

Electricity offers a valuable adjuvant. It may be applied to the conjunctiva over the paralyzed muscle, or to the skin over the closed eyelids, or along the nose and inner margin of the eye. In the former case, the eye must be cocainized, and the weakest possible faradic current applied through a bipolar electrode. The strength of the current must be tested beforehand on the physician's tongue, and the battery must be one giving a perfectly smooth current. In the latter method, the galvanic current is better. The positive electrode should be placed back of the angle of the jaw, or at the nape of the neck, while the negative is moved over the closed lids, and along the inner margin of the eye, and over the skin



of the side of the nose. Its beneficial action here is due not to direct action on the muscle, but to improved nutrition of the parts involved in the central lesion by the reflex stimulation through the skin.

### SPASM OF THE OCULAR MUSCLES.

The different muscles of the eye and its appendages are subject to certain disorders of movements as symptoms of disease of the nervous system. These consist of simple spasm of the external muscles of the eye, nystagmus, conjugate deviation of the head and eyes, and blepharospasm. Simple spasm of the eye muscles gives rise to spasmodic squint. It occurs most frequently in conjunction with lesions producing irritation at the base of the brain. The deviation of the eyes is observed, even when the eyes are at rest in the mid-position. This form of squint is sometimes observed as a reflex from abdominal irritation and in chorea. It very commonly produces diplopia.

Spasm of the ocular muscles also occurs in hysteria, especially during the convulsive seizures. The movements are convergent, never divergent.

Sometimes, the spasm is paroxysmal. In such cases, it comes on suddenly, lasts for a few seconds or minutes, and then as suddenly disappears. The significance of such cases is not understood.

**Nystagmus** consists of rhythmical oscillations of the eyeball. The movements may be lateral, vertical, or rotary, the former being the most common. Their rapidity varies greatly in different cases, and at different times in the same case, the extremes being sixty and two hundred per minute. Usually the movements of the eyes in one direction are more rapid than on the return. In many cases, the nystagmus is observed only when the eyes are turned in certain directions.

The causes of nystagmus are found first, in local conditions of the eyes, and secondly, in diseases of the nervous system. Ocular changes producing this condition are opacities of the cornea and lens, and degenerative diseases of the choroid and retina. These are operative only when occurring in infancy. Nystagmus never arises from errors of refraction. In albinos nystagmus is very common. These defects were combined in no less than four children of one family under my care.

The nervous diseases of which nystagmus may be a symptom are many and varied. They consist mostly of chronic degenerative diseases as disseminated sclerosis, and Friedreich's ataxia, meningitis, meningeal hæmorrhage, thrombosis of the cerebral sinuses, tumor, and local softening. Intracranial tumor is especially liable to give rise to this symptom when affecting the cerebellum. Lesions in the pons, optic thalamus, and corpora quadrigemina have also caused it.

In some cases, the movements may be so slight as to escape observation. In such they can be made more prominent by directing the patient to look slightly upwards.

The pathology of nystagmus is not at all understood. It is known, however, that its presence is proof positive of organic disease.

The prognosis is unfavorable.

**Conjugated Deviation of the Head and Eyes** consists of a movement of the head and eyes to one side, this being accomplished by the external rectus of one eye and the internal rectus of the other and the sternomastoid muscle on the side opposite to the direction of the movement.

It may be either paralytic or spasmodic, its nature being readily recognized by the conditions of other portions of the body. It is very common in the early stages of hemiplegic seizures. That the position of the head in such cases is not due to gravity is shown by the fact that some effort is required to bring the head to the median line, and as soon as it is released, it at once returns to its abnormal position.

The direction of the deviation will vary according to the seat and nature of the lesion. When destructive (paralyzing), a lesion above in the hemispheres causes deviation towards that side and away from the paralysis; the patient tries to look at the lesion, as it has been expressed. With a basilar lesion of the same character the deviation is in the direction of the paralyzed side. When the condition is one of convulsion the opposite to the above statement obtains; in cerebral lesions the deviation is towards the side of the convulsed extremities; and in those of the mesencephalon, towards the side of the lesion. Some apparent exceptions to these observations have been reported.

### THE PUPILS.

Before proceeding with the consideration of the symptomatic value of pupillary disturbances, it is well to present definitions of the terms to be used.

*Mydriasis* is dilatation of the pupil.

*Myosis* is contraction of the pupil.

The qualification *stabile* is applied to mydriasis or myosis when the pupils fail to respond to light; the term *labile* is used when the pupils are movable.

The dilatation of the pupils is effected by contraction of the circular fibres of the iris, and relaxation of the radiating fibres. Contraction of the pupils is produced by the converse of these. It will be readily seen, therefore, that either mydriasis or myosis may result from opposite conditions, paralysis and spasm.

The semeiological aspects of the pupil have been studied very elaborately by Macewen (*American Journal of the Medical Sciences*, July, 1887), and have been ably formulated by him in the following propositions:

"A. (1) When the function of the brain is in abeyance the pupils are in a state of stabile mydriasis.

"(2) This may arise either from temporary suspension or from abolition of function.

"(3) Temporary suspension is illustrated by shock and the effect of some poisons, while the abolition of function is exemplified by extensive laceration and compression of the brain.

"B. (4) When the function of the brain is interfered with by conditions usually included under the term irritation, the pupils are in a state of myosis; sometimes labile, but generally stabile myosis.

"(5) This 'irritation,' or interruption of function, may be seen during certain degrees of cerebral anæmia produced experimentally, and not as a pathological result; certain amount of brain pressure and certain stages of intracranial inflammation.

"(6) These are illustrated in persons who have suddenly lost a considerable quantity of blood (about a fifth of the whole); in the growth of intracranial tumors and the formation of sanguinolent, serous and purulent effusions; when the degree of pressure may be denominated as 'medium,' and at certain periods of meningitis and encephalitis.

"C. (7) The same pathological factors which cause myosis may also cause mydriasis, the degree in which these factors are present being the determining point between the former and the latter, and not merely the particular locus in the brain.

"(8) It is well illustrated by cases where the hæmorrhage is repeated and is finally pushed to syncope; in instrumental pressure, which is gradually increased until it becomes great, such as arises from tumors, blood-clots and inflammatory products.

"D. (9) When the function of one-half of the cerebrum is placed in abeyance by a superficial or cortical lesion, the pupil on the same side of the lesion is placed in a state of stabile mydriasis.

"(10) This is well illustrated in cases of intracranial sanguinolent effusions consequent on injury.

"E. (11) When the function of one-half of the cerebrum is interfered with by some source of cortical irritation, the pupil on the corresponding side to the lesion is in a state of myosis.

"(12) This is illustrated by traumatic and pathological lesions affecting the cortex of the cerebrum.

"F. (13) Hæmorrhage into the pons Varolii when small, causes strongly contracted pupils; but when it is more extensive, involving the gray matter beneath the aqueduct of Sylvius, a state of stabile mydriasis ensues.

"(14) Effusions into the lateral ventricles when small, produce contraction of the pupils, but when the effusion is great, stabile mydriasis ensues.

"(15) Inequality of the pupils indicates a unilateral lesion or lesions.

"(16) When the lesion is cortical and unilateral the pupillary mani-



festations are on the corresponding side. When the basal nerves are affected unilaterally, the pupillary effect is manifested on the same side as the lesion. When the lesion is unilateral and affects the function of the white fibres of the cerebrum, the opposite pupil is generally affected. When the basal ganglia are affected unilaterally, the pupil is sometimes affected on the same side as the lesion, occasionally on the other side. In lesions of the cerebral peduncles, the pupil is affected on the same side as the lesion. Lesions in the corpora quadrigemina affect both pupils, irritation causing contraction, destruction causing dilatation and immobility. Section or destruction of one optic tract causes dilatation of the opposite pupil and blindness of the opposite eye.

“(17) Irritation of the cord, especially of the cilio-spinal axis, produces dilatation of the pupils, while destruction of the cord causes contraction. These effects are generally seen in both pupils, though experimentally at least they may be confined to the same side as the lesion.

“(18) The pupils are affected in the same way by lesions of the sympathetic, though in unilateral lesions it is only the pupil on the same side as the lesion which is affected.

“(19) Speaking generally when myosis is due to a cerebral cause, it indicates the earlier stages of various affections; when due to a spinal lesion, it points to a most serious paralysis, often to a destruction of the part. When mydriasis arises from a cerebral lesion it is generally present in large amount; when due to a spinal affection it indicates irritation of the part.

“Myosis occurs under the following conditions:

“(1) When a bright light acts upon the retina.

“(2) Accommodation for a near object.

“(3) Rotation of the eyeball inwards.

“(4) Local irritation or painful affections of the eyeball.

“(5) Irritation of the oculo-motor nerve.

“(6) Paralysis of sympathetic roots of lenticular ganglion or trunk of sympathetic in the neck. In paralysis of the fifth, there is myosis and inflammation passing on to destruction of the eyeball.

“(7) Paralysis of the cilio-spinal region of the spinal cord. All affections which destroy the cervical spinal marrow and intercept its conductivity, produce congestion of the face and contraction of the pupils. In neurosis, which suspends or diminishes the tone of the sympathetic or spinal axis.

“(8) Encephalic congestion such as obstacle to return of blood in the jugulars; venous congestion due to cardiac causes; active hyperæmia; plethora, fevers, pneumonia, hepatitis, etc.; when animal is suspended by the heels; in early stages of meningitis and encephalitis; in acute mania with marked activity of the cerebral circulation; in chronic

mania pupils are variable, when contracted are said to indicate super-vention of paralytic dementia.

"(9) During sleep; some believe this to be due to the congestion of the cerebral vessels and those of the iris; others to the inward rotation of the eyeball.

"(10) In the early stages of cerebral tumor.

"(11) In small hæmorrhages into the cerebellum. In irritation of the cerebellum, contraction of the pupil on the same as the lesion ensues.

"(12) Electrical stimulation of the angular gyrus frequently causes contraction of the pupil.

"(13) During forced expiration when the eye is at the same time passive. Also generally seen during the period of apnœa in Cheyne-Stokes respiration.

"(14) Convulsions arising from meningo-encephalitis are said to be accompanied by myosis, while in convulsions due to epilepsy, and in epileptiform fits, they are usually accompanied by mydriasis.

"(15) When the pupil contracts on accommodation to a near object, yet does not contract to light, this indicates a lesion situated between the corpora quadrigemina and the oculo-motorius. This affection is known as the Argyll-Robertson symptom. It is seen in locomotor ataxia, and occurs generally in paralysis of the insane.

"(16) During uræmic coma.

"(17) Myotics: physostigmine, nicotine, pilocarpine, muscarine.

"Mydriasis occurs under the following conditions:

"(1) In darkness or subdued light.

"(2) Accommodation for distant objects.

"(3) Rotation of the ball outwards.

"(4) In forced movements discharged from the medulla; vomiting, swallowing, chewing, forced respiration.

"(5) Paralysis of the oculo-motor (accompanied or not by immobility of the eyeball, external strabismus, diplopia, etc.).

"(6) Destruction of the optic; amaurosis. When unilateral, associated movements continue.

"(7) Irritation of the sympathetic; powerful impressions on sensory nerves; strong moral emotions, mental pain, grief, fear; neuralgia of the fifth nerve.

"(8) Irritation of the spinal cord, especially the cilio-spinal region.

"(9) Encephalic anæmia: In all cases where there is reflex contraction of the vessels of the head; then loss of blood from the body is excessive; obstruction of the carotid arteries; in thrombosis of the brain sinuses; dilatation of mesenteric vessels, when extreme; syncope intense cold, rigors; dyscrasias of the blood, convalescence, cachectic conditions; asphyxia, epilepsy, in certain stages of these affections.

"(10) Pressure of cerebrum when great in amount, as from hæmorrhage, neoplasms, etc. In the last stages of meningo-encephalitis.

"(11) In cerebral softening. In acute dementia (œdema of cortex cerebri), observers state that the pupils are invariably dilated (Hutchinson).

"(12) In idiots the pupils are generally dilated.

"(13) During deep inspiration, generally in respiratory period of Cheyne-Stokes breathing.

"(14) Hæmorrhage into centrum ovale and into cerebral peduncles.

"(15) Ferrier produced dilatation of the opposite pupil by destructive lesion of the optic tract in the thalamus, indicative of rupture of the centripetal fibres to the irido-motor nucleus in the floor of the Sylvian aqueduct.

"(16) In hydrophobia there is mydriasis.

"(17) Mydriatics: Atropine, homatropine, duboisine, daturine, hyoscyamine, cocaine. Curare injected subcutaneously in animals induces in one to two hours complete paralysis of the third nerve."



## LESIONS OF THE OPTIC CHIASM, OPTIC TRACT AND VISUAL CENTRES.

The characteristic symptom of a lesion involving the optic commissure or tracts and the cortical visual centres is hemianopsia; and conversely, the existence of hemianopsia indicates beyond the possibility of cavil a lesion involving some portion of the structures above named. According, however, as the lesion involves the visual fibres in this or that portion of their course, so will the characteristics of the hemianopsia or its associated conditions vary. So well understood is this fact, that we find in the clinical investigation of hemianopsia a most beautiful and scientific application of the principles of cerebral localization.

By hemianopsia is meant a blindness in one-half of the visual field of one or both eyes. If it affects a half-field in one eye, it is *unilateral*; if in both eyes, *bilateral*. If the blind half-field is separated from the normal one by a vertical dividing line, we call it *lateral* or *vertical hemianopsia*; if the dividing line is horizontal, *horizontal hemianopsia*. If the blind half-field lies towards the temporal side, it is *temporal hemianopsia*; if both temporal half-fields are involved, *bi-temporal hemianopsia*; if the nasal half-field is destroyed, *nasal hemianopsia*; if the nasal half-field of one eye and the temporal half-field of the other, *homonymous hemianopsia*. The latter term is further qualified by the terms right and left according to the blind side in its relation to the patient. A unilateral lesion posterior to the chiasm always produces blindness of the same named half of each retina. It should be remembered that inasmuch as the rays of light cross on entering the eye, that a temporal hemianopsia is due to a blindness of the nasal half of the retina.

A proper practical understanding of the significance of hemianopsia as a symptom requires a knowledge of the course of the visual tracts in the brain. The accompanying diagram, after Starr, adds to the clearness of a written description. On reaching the optic chiasm, each optic nerve divides into two portions. Those fibres which had come from the temporal half of the retina pass on to the tract of the corresponding side; while those from the nasal half of the retina, decussate, and enter the optic tract on the opposite side. "From the optic chiasm each optic tract passes around the crus cerebri and terminates in the external geniculate body, in the optic thalamus, and in the anterior corpus quadrigeminum, as seen in the diagram. From these three masses of gray matter new fibres start out which issue from the outer side of the optic thalamus, enter the internal capsule, and curve

backward through the occipital lobe, lying on the outer side of the posterior horn of the lateral ventricle, and thus reach the occipital convolutions, ending chiefly in the cuneus." (Starr.)

Hemianopsia is not always recognized by the patient. The latter may be fully aware that his sight is not what it should be; but of the exact nature of the defect he may remain in ignorance. In the case of Patrick H., a soldier shot in the occipital lobes at the battle of Antietam, a case that has become historical, the actual state of vision was not known until several years afterwards. The best method of investigating the condition of the visual field, indeed, the only way for keeping records of the case, and comparing the progress made from time to time, is that with the perimeter. This instrument is not, however, likely to be at the command of the practitioner, and a readier way is necessary. The patient should be seated opposite the physician. One eye should be covered, while the one to be tested should be fixed on a given point of the physician's features, his nose or the pupil of one eye. Whatever point be selected for fixation, the greatest care must be observed to have that point immediately opposite the eye to be tested. If the physician's eyes are utilized, it should be the one opposite named to that of the patient. The physician must, likewise, close his eye not in use, that he may judge his own field of vision correctly. Then, the physician, holding one hand at a considerable distance to one side, and at the same distance from himself and his patient, slowly brings it inwards until it is seen by the latter. He then tests the power of vision in the same way by approaching the hand from another direction, and then from still others. By comparing the patient's statements with the observations of his own eye, he is enabled to state that there is or is not limitation of the visual field. Finding a limitation, he may make a diagram of the same on a black-board. The patient is directed to fix on a given spot, while the chalk is approached from the periphery until seen; that point is marked on the board. The chalk is then approximated from still other directions, and the results noted. By connecting the various points thus obtained, the shape of the field of vision is roughly delineated. During all these investigations, care must be exercised that the patient maintains his gaze on the fixation point.

I have said that the recognition of the seat of lesion in a given case of hemianopsia is a most interesting clinical problem. We may divide lesions into those in front of and those behind the primary optic centres (external geniculate body, pulvinar of the optic thalamus, and the anterior corpora quadrigemina). Cases arising from lesions in front of these parts, *i. e.*, between these ganglia and the chiasm, present the phenomenon known as the hemianopic pupillary inaction. Lesions behind these ganglia do not affect the pupillary reactions. When the hemianopic pupillary inaction is present, light thrown on the blind half of the retina

fails to elicit a pupillary contraction. In applying this test, the greatest care is necessary that the light is thrown on the blind part only; otherwise the observation is valueless. The examination should take place in a darkened room, the source of light should be a small flame, and the ophthalmoscopic mirror should be most carefully handled. The distance at which it is held should be such as to bring the light to the smallest possible point.

When the lesion involves the central portion of the chiasm (Fig. 28, *H*), the decussating fibres are attacked, and bi-temporal hemianopsia results.

Should the lesion increase in size and affect the direct fibres on one side, there will be complete blindness on that side, and temporal hemianopsia on the other.

A lesion attacking the outer fibres of the chiasm (Fig. 28 *N*) produces nasal hemianopsia. Symmetrically placed lesions of this character produce bi-nasal hemianopsia, a *very* rarely observed condition. In one reported case this resulted from atheroma of the circle of Willis.

A lesion of one optic tract causes lateral hemianopsia, the side of the hemianopsia being the side opposite to the lesion. It is possible, however, for a lesion affecting one optic tract to involve only a portion of its fibres. Thus a tumor pressing only upon its outer fibres would cause a nasal hemianopsia in the eye of the corresponding side. According to the direction and force with which the pressure is exerted, very irregular types of hemianopsia may result.

In many cases of hemianopsia arising from disease of the tracts or commissure, there are associated symptoms indicating involvement of certain cranial nerves, or the motor paths, and not infrequently, changes in the ophthalmoscopic appearances.

If the tract is involved at its termination in the ganglia, the hemianopsia is associated with hemianæsthesia; if in the internal capsule, there will probably be hemianæsthesia and hemiplegia; a lesion between the internal capsule and the cortex will cause hemianopsia with or without hemianæsthesia. A lesion of the cortical centres (in the cuneus) gives rise to hemianopsia alone. The absence of associated symptoms here becomes of localizing value.

Aside from the above we may have hemianopsia associated with the most complicated conditions. Thus a complete obstruction of the entire Sylvian artery will give rise to hemianopsia, hemiplegia, and when the left side is affected, aphasia, alexia, etc.

The mode of onset of hemianopsia varies according to the pathological cause. In diseases of the chiasm and optic tracts it is usually gradual, because the lesions appearing in this situation are nearly always of a chronic nature. Cases due to cortical diseases are not infrequently sudden in their appearance because of the possibility of the production



of the trouble by circulatory disturbances (embolism and hæmorrhage). At the same time it must be borne in mind that lesions of any kind may obtain in any portion of the visual tract, each kind impressing upon the case its distinctive features.

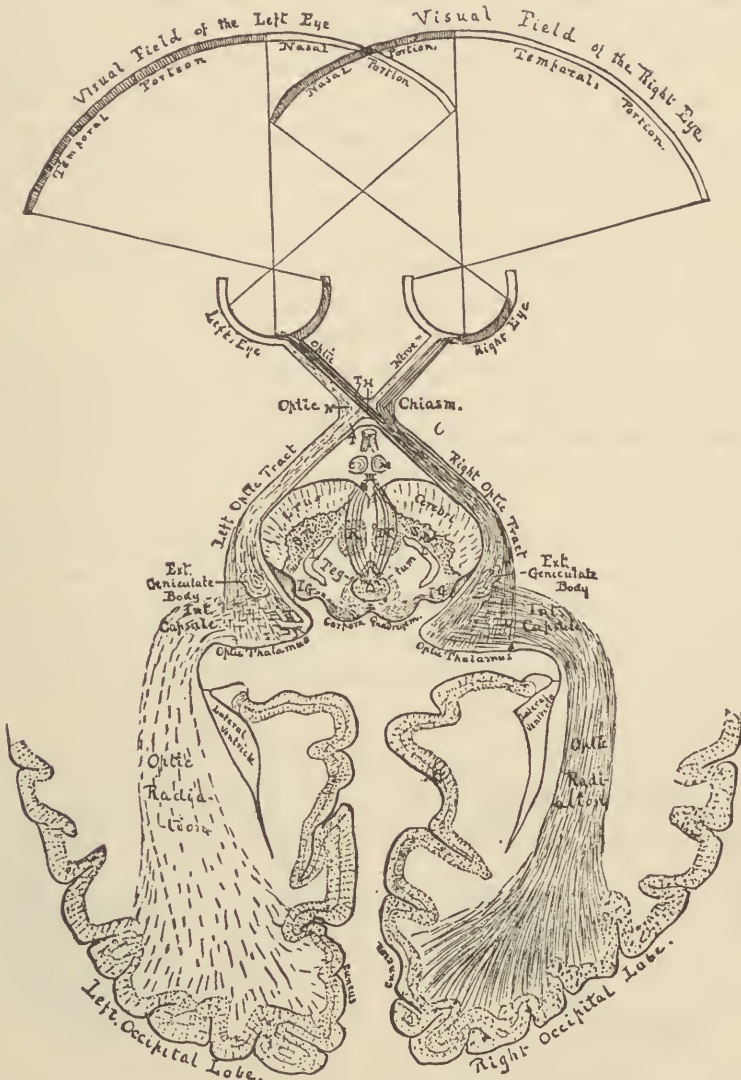


FIG. 28.—THE OPTIC AND VISUAL TRACTS (AFTER STARR). N—LESION CAUSING NASAL HEMIANOPSIA; T—LESION CAUSING TEMPORAL HEMIANOPSIA; H—LESION CAUSING BI-TEMPORAL HEMIANOPSIA. C—LESION OF THE OPTIC TRACT CAUSING LEFT-LATERAL HEMIANOPSIA.

The line of division between the normal and blind fields possesses no fixed character. It may be vertical, slanting, sinuous or indented. It may pass through the fixation point or well within it; it may be vertical

and on a line with the fixation point, but when within a few degrees of the latter, it very often curves towards the blind half, leaving the fixation point within the seeing area. The preservation of central vision bears no relation whatever to the seat of the lesion; it probably depends upon individual peculiarities in the decussation, the fixation point on the retina receiving fibres from both sides.

This phenomenon is susceptible, however, of another explanation, *i. e.*, the existence of a centre for central vision outside of the half-vision centres in the cuneus. Four cases bearing on this point have now been recorded. In each there was a first attack of hemianopsia with preservation of central vision for a distance of from three to eight degrees about the fixation point. A second attack of hemianopsia destroying the remaining half-fields occurring, the central vision was still retained, although its acuity was considerably impaired.

The course pursued by a hemianopsia is not a typical one. In some cases the defect remains stationary for years. In others it exhibits very great variations from time to time. Oppenheimer has said that an oscillating bi-temporal hemianopsia is almost always of syphilitic origin. In disease of one optic tract it is not usual for the defect to extend to the uninvolved half-fields. Still there may be concentric limitation of the latter by reason of the tract lesion setting up inflammation of the optic nerve-fibres beyond the commissure. In hemianopsia from disease of the occipital lobe the blind half-field may extend to a line vertical with the fixation point, but never beyond it. The fact that it has done so is significant of extension of disease to the opposite side of the brain.

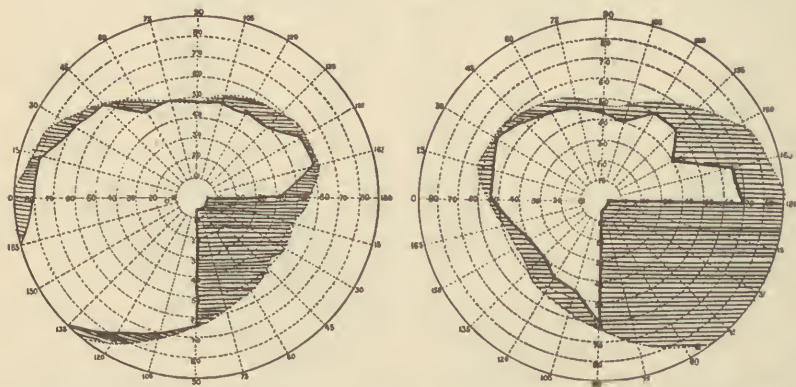


FIG. 29.—HOMONYMOUS HEMIANOPSIA AFFECTING THE LOWER QUADRANTS FROM FIBRO-SARCOMA OF THE CUNEUS.

The blindness is not always symmetrically distributed, when the lesion is in the tract or commissure. Indeed, irregularity in the defect is here the rule rather than the exception. In disease of the occipital lobe, on the other hand, symmetry of defect is the rule. The latter symptom is therefore indicative, though not conclusively, of cortical

lesion. I here append the perimeter charts of a case of lateral homonymous hemianopsia in which the lower quadrants are defective. Such cases are not uncommon. Usually they result from disease of the cuneus, the fibres from the lower half of the cuneus going to the upper half of the retina, and *vice versa*. It is believed, and with good reason, too, that each portion of the retina finds its representation in a special portion of the cuneus. This fact is of importance in the study of scotomata, especially when the latter are symmetrical.

When hemianopsia has lasted for any length of time, it is apt to remain permanent. The only possible exceptions are those arising from syphilitic disease which has not acted destructively on the nerve elements.

A functional hemianopsia has been claimed. There can be no doubt of its existence if we refer only to transitory attacks of this symptom in conjunction with migraine, or similar attacks unassociated with cephalic pain. These latter are probably abortive migrainous seizures. Dana has referred to lithæmia and gout as causes of hemianopsia, but I should feel very doubtful of their ability to produce such a condition without first giving rise to structural alterations in the visual fibres.

Hemiopic hallucinations have been described by Seguin, Peterson, Putzel and others. By the former author it is stated that in two out of nine cases of ordinary homonymous hemianopsia these visual hallucinations appeared in the subsequently blind field prior to the onset of the blindness. In Peterson's cases the visual disturbances existed in conjunction with mental trouble and without any associated blindness.

In certain rare cases the blindness in the half-field takes the form of a network, the remaining spots giving more or less acute vision.

Horizontal hemianopsia is nearly always due to retinal disease. Nothnagel has, however, reported a case in which he believed the trouble lay in the occipital lobes.

The treatment of hemianopsia must be based entirely upon the associated conditions. It cannot be treated *per se*.



## AFFECTION OF THE FIFTH NERVE.

The fifth pair of nerves possesses both sensory and motor functions, the motor branches supplying the muscles of mastication with the exception of the buccinator, and the sensory branches furnishing sensation to the face. Symptoms arising from diseases of these nerves will necessarily differ according to the special portion affected. We may have spasm, paralysis, pain, paræsthesia or anæsthesia, or any combination of motor and sensory phenomena.

### PARALYSIS.

**Etiology.**—Paralysis of the motor branch of the fifth nerve may arise from quite a variety of conditions. It may be due to disease within the pons (generally some focal lesion other than degeneration), certain diseases at the base of the brain, as tumors, meningitis, etc., and disease in the course of the nerve itself. Of the lesions in the course of the nerve itself we have neuritis and traumatism. The former almost always occurs as secondary to other morbid processes, although occasionally it results from exposure to cold, syphilis, or gout. The form of neuritis which is associated with or lies at the bottom of herpes zoster is very common in this nerve. Traumatic lesions of the trigeminus are rare as complicating fracture of the skull or direct blows, but occur occasionally as the result of gunshot wounds through the mouth or nasal cavity. These same causes affecting the sensory fibres of the nerve produce anæsthesia, etc.

**Symptoms.**—Paralysis of the muscles of mastication requires a little care for its detection when the loss of power is slight. The patient should be directed to close his jaws firmly. The examiner at the same time places his fingers over the contracting muscles on both sides, and notices their relative powers. In mild cases he may note only that the muscles on the diseased side contract somewhat more slowly than those on the other. The temporal, masseter and pterygoid muscles are paralyzed. That of the former two is shown by deficient power of mastication on the affected side; that of the pterygoids by inability to move the lower jaw toward the healthy side. Masticatory paralysis is very frequently bilateral. When due to disease of the nerve itself, the characteristic reaction of degeneration is present. Subsequently the muscles waste. This late atrophy was well illustrated in a case seen by me a number of years ago. The patient, a woman, while singing in church attempted to gape, and felt something snap back of the jaw on one side. She next noticed

a wasting of that side of the face, and when I saw her, the temporal and maxillary fossæ were devoid of muscular tissue. Paralysis of the muscles of mastication on one side may be due to a cortical lesion. Such cases, are, however, exceedingly rare.

### SPASM.

**Etiology.**—Spasm of the muscles supplied by the fifth nerve may be either tonic or clonic. Both varieties occur as parts of general convulsive phenomena, rarely, on the other hand, as the only symptom. Tonic spasm, generally spoken of as trismus or lockjaw, is a common condition in tetanus, and is occasionally of hysterical origin. It sometimes arises from reflex irritation, as from the teeth, especially from disease of the last molar. It may likewise occur in consequence of organic disease of the pons, as tumor. In the latter case the spasm is generally bilateral, though often more prominent on one side than the other, even when, as is usually the case, the lesion is unilateral. It has also been produced by exposure to cold, and by intestinal disorders. It sometimes occurs in the new-born, in which case it is called trismus nascentium. The cause of this infantile trismus is subject to considerable difference of opinion. One view attributes it to infection from the umbilicus and another to pressure and displacement of the cranial bones. Cases apparently cured by attention to posture have been reported.

Clonic spasms of the masticatory muscles are very uncommon excepting as part of ordinary convulsive seizures. Single clonic spasms, that is, a sudden jerking closure of the jaws, are sometimes observed in choreiform conditions. They rarely occur as an isolated symptom. They have been reported as taking place during sleep and causing biting of the tongue.

Occasionally one meets with cases of involuntary chewing motion, the precise pathological origin of which is unknown.

### SENSORY DISTURBANCES.

**Paræsthesia.**—Morbid sensations not amounting to pain, and not partaking of the character of anæsthesia are rather frequently observed in the area of distribution of the fifth nerve. They are almost always symptomatic of neurasthenia, or some functional nervous disorder. They are sometimes found in the anæmic.

**Anæsthesia.**—The most common cause of anæsthesia of the fifth nerve is syphilitic disease of the brain. It may occur in hysteria in conjunction with anæsthesia, of other parts of the body. The parts supplied by the fifth nerve lose their sensation. Preceding the anæsthesia, there may be some irritative phenomena partaking of the character of neuralgia. Tender points along the course of the nerve are often observed. In other instances, the anæsthesia is the first abnormal con-

dition. It is spread over the face and mucous membranes of the eye, nose and mouth. The tongue on the affected side becomes much furred, probably because the patient does not chew on that side, and thus the coating is permitted to accumulate. After a time the sense of smell is lost or impaired, not, however, from any involvement of the special nerve for that sense, but by reason of the defective nasal secretion and the trophic changes in the nasal mucous membrane. Taste is lost on the affected side of the anterior two-thirds of the tongue. Trophic phenomena are present. These consist of lessening of the conjunctival and buccal secretions, ulcerations of the mouth and neuro-paralytic ophthalmia. The precise nature of this last-named condition is a subject of dispute. Some believe it to be the logical result of the dryness of the conjunctiva. The weight of evidence favors the view that it is a true trophic phenomenon. It is quite a serious condition, often leading to panophthalmitis and destruction of the eye. The cheek is not infrequently bitten during mastication, and being insensitive, the injury very often escapes notice. An ulceration is thus produced, and this proves very obstinate to treatment. Occasionally the teeth become loose. In other cases herpes zoster appears, and in the aged gives rise to severe and long-standing suffering.

**The Diagnosis** of the different lesions of the fifth pair of nerves is usually a comparatively easy matter. In obscure cases a study of the anatomy and physiology of the parts should be made. Observation has taught that the different branches of the trigeminus are liable to lesions in special portions of their course. When all parts of the nerve are affected, then the lesion must be in the Gasserian ganglion or in the brain; when the supraorbital branch or first division only is affected, the disease is usually situated at the sphenoidal fissure or in the orbit; when the second division, in the spheno-maxillary fossa or the superior maxillary bone; when the second and third together, in the sphenoid bone or the spheno-maxillary fossa.

The association of paralysis of other parts with trigeminal disturbance offers suggestions for localization of the lesion. If the oculomotor nerves are involved along with the fifth, then the lesion must be at the side of the pons; if only the first division of the fifth is affected, in the middle fossa of the skull. The significance of the association of fifth nerve paralysis with hemiplegia varies according to the mode of onset; if sudden, it is due to disease within the pons; if of gradual onset, to disease within or out of the pons. "Inability to move the eyes towards the side of the lesion is evidence of disease within the pons."

**The Prognosis** will depend entirely upon the nature of the pathological condition in each case, *e. g.*, that of isolated spasm of the muscles supplied by their nerve, is almost always good; when symptomatic of other conditions, it depends on the nature of the primary affection.



**The Treatment** must be conducted on general principles. If the cause of trouble can be removed, it must receive attention. Anæsthesia, paræsthesia, paralysis spasm must be treated by remedies prescribed according to indications given in the *materia medica* or in paralysis, anæsthesia, etc., elsewhere.

Electrical treatment is indicated. For the anæsthesia the faradic brush should be employed, making the current sufficiently strong to produce a well-marked sensation. For the paralysis, stimulation of the affected muscles and nerves by the interrupted galvanic or faradic current is of value.

### TRIGEMINAL NEURALGIA.

**Nomenclature.**—Prosopalgia; facial neuralgia.

All cases of neuralgia affecting the trigeminal nerves are, by no means, of the same origin. Clinically they may be divided into two classes: (1) the simple, sympathetic or reflex; and (2) the essential or *tic douloureux*. The former are met with by all odds the more frequently; indeed, they constitute a large class of the cases coming before the practitioner in his daily work. I shall therefore consider these first.

**Etiology.**—All agencies tending to constitutional deterioration, as poor nutrition, anæmia, overwork, frequent child-bearing, neurasthenia, etc., may be rated 'as causes of simple facial neuralgia. Women being the more exposed to these are naturally the more frequently affected. Among the local or reflex causes dental troubles take the first place. Errors of refraction are not infrequently a cause, although they are more likely to produce headache, rather than neuralgic pain in the face. Nasal disease is an occasional cause. Other neuralgias are due to some specific influence, notably to malarial infection, rheumatism, gout, syphilis, and diabetes.

**Symptomatology.**—The characteristic symptom in neuralgia of the fifth pair of nerves is pain in their area of distribution. This pain is usually of a sharp lancinating character, although it may be dull and subdued. Its duration varies greatly in different cases, from a few hours to many days. In severe and long-lasting cases, it may be associated with some of the trophic changes to be mentioned as accompanying *tic douloureux*. The pain is usually limited to the area supplied by one branch of the trigeminus. Its seat bears no definite relation to the exciting cause. While in the case of neuralgia arising from diseased teeth, the pain is usually in the vicinity of the offending part, still it may occur anywhere else. Thus I have seen pain in the lower jaw caused by a bad tooth in the upper.

Associated with the pain is a feeling of pressure or tension, and frequently sensation of numbness or formication in the affected parts. There may be either hyperæsthesia or anæsthesia of the skin over the

painful area. The former is said to occur in recent, the latter in chronic cases of neuralgia. At times we have sympathetic involvement of the facial, in which case motor phenomena, such as blepharospasmus and twitching of the muscles of expression, are observed. The special senses may be affected. Subjective noises are heard in the ears. Flashes of light may appear before the eyes; vision may be dulled or photophobia may manifest itself, together with profuse lachrymation, any exposure to light greatly intensifying the pain. Alterations in the sense of taste may be exhibited and the salivary secretion increased.

**Diagnosis.**—The diagnosis of a given case of neuralgia consists in determining the actual cause and the pathological changes present. In recent cases dependent upon cold or local irritation, this is an easy matter. In chronic cases a most careful clinical study of the patient is necessary. In examining the mouth it must be borne in mind that dental caries is not the only disease of the teeth which may cause pain. On the contrary, inflamed or congested nerve pulp, secondary dentine in the pulp cavity, confined gas from decomposed pulp, exposure of the sensitive peridental membrane, pyorrhœa alveolaris, and other conditions too numerous to mention, may all excite neuralgia, and must be carefully sought for. In doubtful cases the patient should be recommended to a dental *expert* for examination. While, as I have already stated, there is no definite relation between the seat of the pain and the site of the affected tooth, there is some general rule for our guidance. When the pain is situated in the eyeball, which is not infrequently the case, the offending tooth may be any of those in the upper jaw. When the pain is in the upper part of the head or in the temporal region, the diseased tooth is generally in the upper jaw, and in the back part of the mouth. When the pain is referred to the ear, we should look to the posterior teeth of the inferior maxilla. The diagnosis of errors of refraction, and of such constitutional conditions, as neurasthenia, anæmia, rheumatism, gout, etc., is self-evident, and requires no elaboration here.

**Prognosis.**—The prognosis in simple facial neuralgia depends upon the readiness with which the cause is removed. Usually the affection lasts as long as the exciting cause. In the vast majority of cases this is removable, and the patient readily cured. Other things being equal, the prognosis is more favorable in the young than in those of advanced years. Many cases occurring late in life when there is nerve as well as general tissue degeneration, are very intractable.

### TIC DOULOUREUX.

**Nomenclature.**—Fothergill's neuralgia; epileptiform neuralgia.

This is a very different affection from the form of neuralgia just considered. Its etiology is as yet very imperfectly understood. It is, however, believed to be a disease of the degenerative period of life, the

majority, indeed nearly all, of the cases occurring in patients of advanced years. No doubt exposure, overwork, and malnutrition, may act as causes by increasing the tendency to systemic degeneration.

**Pathology and Morbid Anatomy.**—This is largely a matter of speculation. Carefully conducted autopsies have disclosed important changes of varied character. In some cases cranial and intracranial disease has been discovered. Dana calls attention to a low grade of neuritis or nerve degeneration dependent upon an obliterative inflammation of the arteries supplying the nerve.

**Symptomatology.**—Tic douloureux was called by Trousseau epileptiform neuralgia by reason of the peculiar mode of onset and disappearance of the pains. These are of a sharp darting character, coming without warning in paroxysms, which last from a few seconds to a few minutes, and then disappear as suddenly as they came. These seizures may recur many times in the course of twenty-four hours, or they may disappear for days and weeks at a time. The pain is usually limited to one of the branches of the trigeminus; certainly in the early stages. Later other branches become sympathetically or organically affected. During the paroxysm the patient puts his hands to the affected part and exerts great pressure. Indeed this frequently applied pressure is often the cause of local changes in the parts. The face becomes flattened, and the hair rubbed off. During the paroxysm there may be severe local twitchings, lachrymation, coryza, or salivation. The pains may be re-excited by slight irritations, as a breath of cold air, eating, talking, or putting out the tongue.

Trophic phenomena are frequently observed in old cases. Vasomotor paralysis may occur, and this gives rise to local heat and vascular engorgement. From this cause the lips may even become thickened and in a condition of hyperplasia. In many cases changes in the hair and skin are observed. The latter becomes glossy. Amblyopia and psychical changes may ensue. In two cases I have observed cataract on the affected side, the other eye in each case being in apparently good condition.

In many cases, and this remark applies also to the simple varieties of neuralgia, there are so-called sensitive spots. These will be found at the points where branches of the trigeminus emerge from their bony canals, or become superficial by reason of passing through one or another fascia or muscle. They are as follows: In *supraorbital neuralgia*, the supraorbital over the foramen of that name; a palpebral, over the outer part of the upper lid, a nasal over the nasal bone; in *infraorbital neuralgia*, over the infraorbital foramen, the nasal, on the side of the nose, molar over the prominence of the cheek, and gingival along the gums; in *inferior maxillary neuralgia*, the inferior dental over the point of emergence of that name from its foramen; the temporal in the posterior part of the temple, and the parietal over the parietal eminence.



**Diagnosis.**—The symptomatology of tic douloureux is so distinctively characteristic that diagnostic error is unlikely. Some few cases probably depend upon organic causes. These latter can only be recognized by thorough clinical investigation. It may be desirable to determine whether the pain arises from deep or superficial disease. Here the suggestion first offered by Starr is exceedingly valuable. A few minims of a four per cent. solution of cocaine are injected about one of the branches of the affected nerve. If the disease is peripheral to the seat of injection, the pain will disappear. If central, it will be uninfluenced.

I have known the lightning pains of ataxia to be limited to the trigeminus. The recognition of the true ailment is here made easy by the discovery of characteristic ataxic symptoms.

**Prognosis.**—The prognosis of tic douloureux is very unfavorable. Medicinal means cure but a limited number of cases. Surgical measures generally bring relief, which, unfortunately, continues for but a few years in the majority of cases. A respectable minority of cases are cured.

**Treatment.**—The proper treatment of facial neuralgia includes the thorough treatment of the patient. All constitutional dyscrasiæ must be taken into consideration and treated hygienically and medicinally. Indeed, attention to them is of far more importance than treatment of the pain itself. In fact the physician should forget the pain except as evidence of disease—a prayer of the system for relief. Every possible source of reflex irritation must be investigated and corrected. The constitutional condition of the patient must be thoroughly studied, and the diet and general habits directed accordingly. Routinism in practice is certainly most pernicious, and is a certain barrier to the successful treatment of neuralgia.

With remedies as with hygiene, routinism is out of the question. No drug is a specific. In many instances it is desirable to prescribe on constitutional conditions only, as in the case of neuralgias dependent upon gout, rheumatism, lithæmia, anæmia, etc. Even then individualization is essential. Certain medicines present strong claims for use by their pathogenetic effects, and have been administered with good results by reason of certain general indications.

*Aconite* is a remedy adapted to recent cases of neuralgia, attended by local congestion and rheumatic phenomena. Usually the trouble has arisen from exposure to dry cold winds. The pain is generally associated with tingling in the affected parts.

*Belladonna* may be administered in either acute or chronic cases. The pain is of sudden onset, and may disappear as suddenly as it came. The attendant phenomena are redness of the conjunctiva, lachrymation, and bright red flushing of the face.

*Arsenicum* is undoubtedly one of the most efficient remedies in the treatment of purely nervous neuralgiæ. The pains are often of

burning character, and are worse at night, particularly after midnight. They are prone to be associated with an intense degree of restlessness. Arsenicum is likewise adapted to neuralgiæ of malarial origin. Although not often followed by brilliant results, it is probably our most efficient remedy in tic douloureux. In malarial neuralgia, it is probably most efficient given in the form of Fowler's solution.

*Argentum metallicum* is indicated in purely nervous cases. The pains increase gradually until they reach their acme, and then suddenly cease.

*Actea racemosa* should be given in debilitated women with uterine and ovarian disease. It may likewise be called for in rheumatic cases. Special indications are supraorbital neuralgia and sensation at the top of the head as if it would fly off.

*Chelidonium* is regarded by Hughes as a most efficient remedy for right-sided supraorbital neuralgia. Ferinat regards it as a wonderful remedy for neuralgia in the right temple and eyebrow. Farrington claims, however, that it will not bring relief unless the hepatic symptoms of the drug are present.

*Magnesia phos.* has effected a number of cures on the modality, relief of pain by hot applications. The pain is worse on the right side, and is apt to recur regularly at night.

*Sulphur* and *phosphorus* have accomplished considerable in cases of tic. They must be given, however, in strong doses.

*Kalmia* is called for in supraorbital neuralgia associated with ptosis. The pains are of burning character and are worse on the right side.

*Colchicum* has left-sided prosopalgia. The pains are associated with paralytic weakness of the muscles.

*Cedron* has frequently relieved supraorbital neuralgia, characterized by recurrence at the same hour each day. The pains are of intense burning character, and are worse on the left side.

The *cinchona* case is likewise characterized by its periodicity. Malarial intoxication is sometimes at the bottom of the trouble. The patient is anæmic and debilitated.

*Mezereum* has neuralgia over the cheek-bone, or over the left eye. The pains are followed by numbness. It is especially indicated in neuralgia following zona.

*Platina* has numbness and tingling in the affected parts. The pains increase and decrease gradually.

*Spigelia* is the favorite remedy of Bæhr. The prosopalgia is left-sided, and the pains are of a severe, burning, sticking character. The pains seem to come and go with the sun. The sufferings are greatly intensified by slight external influences.

*Gelsemium* is a valuable remedy in cases attended by paralytic phenomena, or when the circulatory condition of this remedy is present. It may be given in large doses, if the pain fails to yield to ordinary ones.

There are some cases that unfortunately fail to yield to the before-mentioned medicines. It seems that they are well nigh incurable. For their relief, we are obliged to resort to palliative medication, or to surgical measures. Speaking first of the former, let me protest in the strongest possible terms against the use of morphia in this connection. It is true that Trousseau has advocated the administration of this drug in incredibly large doses for the cure of tic douloureux. It is inconceivable to me that the drug thus administered can exert a true, curative effect; and, what is more, its prolonged use must introduce a new phase into the case, that of chronic morphinism. In the ordinarily severe prosopalgias, its use is none the less deleterious. If one is obliged to resort to an analgesic, it should be acetanilid, exalgin, or phenacetin, each in its suitable dose. Under no circumstance should any of these be exhibited recklessly.

Local measures have occasionally brought great relief. Prominent among these stands the applications of drugs which refrigerate the parts. Rhigolene and chloride of methyl, especially the latter, have gained a good reputation in this connection, and have cured some cases that have resisted all other measures. Chloride of methyl, when sprayed on the parts, freezes them promptly. It produces a refrigeration of  $-9.4^{\circ}$  F. The tissues become hard like wood under its influence. Considerable pain is produced for a few seconds. Under no circumstances should the spray be directed against any one spot for more than three or four seconds.

Of late years *aconitine* has come to the front as a remedy for tic douloureux. In using this drug the greatest care should be exercised to secure an efficient preparation. That of Duquesnel is considered the best. The initial dose of the drug should be one two-hundredth of a grain three or four times daily. This dose should be gradually increased until relief is obtained, or until the physiological effects of the medicine are produced. Seguin, in particular, has reported some remarkable results from aconitine. He lays great stress, however, on the importance of an efficient dosage.

All remedies failing, we have certain surgical measures offered to us. These are nerve stretching, nerve resection, nerve avulsion, excision of the Gasserian ganglion, and ligation of the common carotid. All of these procedures have been followed by great amelioration in individual cases. It is very rarely, however, that the result is a permanent one. Some authorities strongly urge the claims of stretching; for, say they, when the disease returns, the operation may be repeated an indefinite number of times. Resection of the nerve affected offers better prospects, however. The portion of the nerve removed should be as extensive as possible. Avulsion seems to be decidedly unscientific, if not really brutal. In several cases, where these operations have failed to cure



permanently, removal of the Gasserian ganglion has been practised with good results. Although but few cases have died under the operation, its great danger must not be forgotten. When all operations on the nervous apparatus have failed, we may lastly resort to ligation of the common carotid. This has been performed but a few times for tic douloureux. Andrews, of Chicago, proposes that when the pain returns after resection of the nerve, that the cicatrix from the first operation be opened and stretched, he believing in this way he gets some mechanical effect on the nerve fibres deep within the bony canal. He reports two cases in which this was done with excellent results.

Literature teems with many other suggestions respecting the treatment of facial neuralgia, but they are for the most part unreliable, or have the indorsement of insufficient authority.

## AFFECTIONS OF THE SEVENTH PAIR.

### (a) FACIAL SPASM.

Strictly speaking, facial spasm should be regarded as but a symptom, and not a distinct clinical entity. Inasmuch as there are observed quite a number of cases in which we are unable to recognize a definite pathological cause for the trouble, we are forced to consider an idiopathic facial spasm. This is likewise spoken of as mimic spasm, convulsive tic, and painless tic. As a secondary disorder it may accompany or follow organic brain lesions. It has been noted in old paralyses of the facial muscles, and is occasionally a symptom of quite a variety of brain lesions, especially those affecting the cortical face centres or irritating the facial nerve in its intra-cranial course. Cases apparently of reflex origin are occasionally observed, the irritation being located within the area of distribution of the fifth pair of nerves.

The idiopathic variety occurs in subjects of advanced or middle life possessing a neurotic constitution. It has been attributed to shock, injuries, and exposure. I believe that it may be occasioned by excessive use of tobacco.

**Symptomatology.**—The onset of the spasm is usually very gradual. So slight is the difficulty at first that it rarely gives the patient any concern. The orbicularis palpebrarum and the zygomatici muscles are usually the first attacked. The spasm extends until possibly all the lower facial muscles are involved. The spasm is usually clonic in character, and takes the form of numerous lightning-like twitches for a second or two, when there is a short rest. Then the convulsive phenomena are repeated. Thus the condition continues day after day, interrupted only by sleep. It may be intensified from time to time by nervous excitement and other emotional influences, as well as by causes which lower the tone of the general health. It is unattended by pain or other evidence of ill-health.

Two varieties of local facial spasm are comparatively common, namely blepharospasm and nictitating spasm. The former consists of a tonic spasm of the orbicularis palpebrarum, of variable duration, and coming at long or short intervals. The spasm may be usually checked temporarily by pressure exerted on certain points about the orbit, notably the point of escape of the supraorbital nerve.

Nictitating spasm consists of winking movements of the lids. It is very common.

**Diagnosis.**—The only diagnostic difficulty is encountered in determining the cause of the spasm. Facial spasm from organic disease of the brain will nearly always be accompanied by other symptoms. Hysterical spasm of the face is usually tonic.

**Prognosis.**—This is very unfavorable as regards cure. Life, of course, is not endangered. The spasm has frequently been known to disappear during the course of acute diseases, and has disappeared permanently after typhoid fever.

**Treatment.**—Galvanism promises more in the treatment of facial spasm than does any other remedy. Care should be exercised in its application. The position of the negative pole is probably a matter of indifference, authors having individual preferences as to locating it. The positive electrode should be placed over sensitive points when they can be found, or failing in that, over the motor points of the affected muscles. Dana recommends an additional application of the current over the occiput and the cortical facial areas for some little time.

Agencies liable to act as reflex causes must be carefully investigated. The teeth and the refraction should be examined, though with faint hope of thereby obtaining an efficient treatment. It is possible for irritation at a distant point to be at the bottom of the trouble. Great conservatism is advisable in acting upon the results of these examinations, lest heroic and deforming operation be undertaken without effecting a cure.

Weir Mitchell in all his experience has cured but one case, and that one by freezing the cutaneous surface of the cheek by rhigolene spray. The applications were made daily for awhile, after which the seances were less frequent.

Surgical measures are sometimes necessary. In blepharospasm, division of the orbicularis has been performed. Excision of the nerve has been recommended, but is altogether too heroic a procedure. Sinkler reports one case of long standing in which nerve stretching was performed with successful result. The affected side of the face was paralyzed for some little time after the operation, but it afterwards regained its functions.

The remedies that offer the best prospects are *hyoscyamus*, *ignatia*, *belladonna*, *hypericum*, *arnica*, *agaricus*, *agaricine*, *mercurius* and *nux vomica*.

## (b) FACIAL PARALYSIS.

**Etiology.**—Facial paralysis of peripheral origin arises most frequently from cold, especially from exposure to strong cold winds in persons of a rheumatic diathesis. It may also follow various mechanical injuries, such as prolonged pressure by sleeping with the head on the closed hand, and section of the nerve or one of its branches during sur-



gical operations about the face. Very rarely it may arise from the pressure of various tumors, notably those affecting the parotid gland, and still more rarely by enlargement of the cervical lymphatics. As a complication of suppurative middle ear disease it is not very uncommon. The only case of this kind coming under my care made a good recovery. Caries or fracture of the petrous portion of the temporal bone, periostitis or hæmorrhage affecting the aqueductus Fallopii, may likewise be causes of facial palsy. I believe that it is a more frequent accident from forceps delivery than is usually claimed. I myself have seen two such in my limited obstetrical experience. One of these cases recovered completely within twelve hours, the other inside of a week. It is very probable that such mild cases often escape observation.

**Symptomatology.**—The onset of facial paralysis will be sudden or gradual according to the influences which have given rise to the trouble. When it has followed exposure to strong cold winds, its advent will be rapid; but where it is the result of pressure of tumors on the facial nerve, its invasion is more gradual. The appearance of the face is characteristic. The paralyzed side is smooth, flaccid, and expressionless. Because of the involvement of the occipito-frontalis and corrugator supercilii, all furrows and wrinkles are obliterated from the brow. The orbicularis palpebrarum is paralyzed, so that the eye remains wide open, even during sleep. The conjunctiva, no longer protected from dust and foreign bodies floating in the air, may become inflamed and painful. Paralysis of Horner's muscles, which is but a part of the orbicularis palpebrarum, prevents the flow of tears into the punctum, so that they overflow the lid and cheek and excoriate the skin with which they come in contact. On asking the patient to close the eyes, the eyeball rolls upwards, and the upper lid is raised still higher. The tissues of the face hanging loose and flaccid draw down the lower lid, producing ectropion, which may in long-standing cases become permanent unless remedied by operation. The buccinator is paralyzed. During the act of chewing, the food constantly slips from between the teeth and lodges in the space between the gums and the cheek, to effect its removal from which situation the patient often calls in the assistance of his finger and tongue. The mouth is drawn obliquely towards the healthy side, while the angle of the paralyzed side is depressed. The paralysis of one half of the orbicularis oris interferes with the prehension of food. The patient cannot purse up the lips as in the act of whistling. His pronunciation of words containing labials is indistinct. Saliva dribbles from the paralyzed side. The nostril on the paralyzed side falls in owing to involvement of the dilator nasi. The sense of smell is apt to be impaired, not because of any involvement of the nerves directly concerned in olfaction, but because of paralysis of the compressor nasi and dilator nasi muscles, this condition interfering with the inhalation of odoriferous particles by

the corresponding side. Further than this the paralysis of Horner's muscle prevents the carrying of the tears through the lachrymal duct into the nose, so that its mucous membrane is unnaturally dry. This also interferes with the proper performance of the function of the olfactory nerve. The above-named symptoms are those commonly observed when the facial nerve is affected after it has emerged from the stylo-mastoid foramen. When the nerve is affected within the bony canal, other symptoms than those above enumerated make their appearance. To appreciate these it will be necessary to give a brief résumé of the anatomy of the facial nerve and its branches. The facial nerve finds its apparent origin in the groove between the olivary and restiform bodies of the medulla oblongata. In leaving the cranial cavity, it enters the internal auditory meatus in company with the auditory nerve. At the bottom of the internal auditory meatus it enters the aqueductus Fallopii, through which it passes, making its exit at the stylo-mastoid foramen. While in the latter bony canal, the nerve gives off several branches of importance. First we find an enlargement of the nerve known as the geniculate ganglion, from which spring the great, small and external superficial petrosal nerves. The first named of these connects with Meckel's ganglion, and supplies the levator palati and azygos uvulæ muscles. The small superficial petrosal nerve connects with the otic ganglion and with the tympanic branch of the glossopharyngeal; the last named connects with the plexus on the middle meningeal artery. Symptoms arising from involvement of these latter two branches have, according to Althaus, not been observed. The next branch is to the stapedius. A little further on in its course, the chorda tympani is given off. This last is the nerve of special sense for the anterior two-thirds of the tongue. Then just before the nerve emerges from the stylo-mastoid foramen, it gives off a branch which supplies the muscles of the external ear. If the paralyzing lesion should involve a point just above the giving off of this last-mentioned branch, there will be, in addition to the symptoms already detailed, paralysis of the muscles of the external ear. If the nerve be involved above the origin of the chorda tympani, but below that of the branch to the stapelius, there will be loss of taste on the corresponding lateral half of the anterior two-thirds of the tongue, together with more or less dryness of the affected side of the mouth. Some observers have ascribed this dryness of the mouth to the fact that on the paralyzed side the lips are separated, and that the admission of the increased quantity of air causes a rapid evaporation of the saliva. The experiments of Schiff have shown that section of the chorda tympani is followed by a diminution in the flow of saliva. To the dryness of the mouth has been ascribed the loss of taste in the anterior two-thirds of the tongue. Taste may, however, be impaired in cases in which there is no dryness. When the nerve is affected above the origin of the branch

to the stapedius, there is a peculiar hyperacusis of hearing, which has been attributed by some observers to the unantagonized action of the tensor tympani, but by Brown-Sequard, to vaso-motor spasm of the internal ear. If the nerve be affected above the geniculate ganglion, the elevator palati and azygos uvulæ will be paralyzed. On examining the throat, the palate no longer presents a symmetrically arched arrangement, but a depression or drooping of the arch on the affected side. Now if the nerve be affected before it enters the aqueductus Fallopii, all the symptoms above enumerated, with the exception of the loss of the sense of taste, will be present.

The behavior of the affected muscles under the stimulus of the galvanic and faradic currents is of great importance. For the first few days the response of the muscles to either current is normal, or nearly so. If there is any alteration, it is generally a more ready response to the stimulus of galvanism. In some cases this may be the only change present at any time. In others, however, faradic contractility rapidly diminishes, so that at the end of a very short period, even in a few days in severe cases, it may be entirely abolished. At the same time the muscles exhibit a remarkably increased irritability to galvanic applications, the resulting contractions being slow and wavy, and not sharp and quick, as in health. Briefly, the reaction of degeneration is present in its typical form.

Facial paralysis may occasionally occur on both sides at once. It is then known as facial diplegia. Such a condition is usually the result of intracranial disease. The eyes stand wide open, the face is devoid of expression, and in fact, all the symptoms of facial monoplegia exist on both sides.

**Diagnosis.**—In severe cases of facial paralysis no difficulty will present itself in diagnosis. In mild cases, or in infants, the physician may be puzzled at first; but careful observation of the movements of the lips and muscles of expression will guard against error. The best guides in infancy are probably found in the failure on the part of the little patient to properly grasp the nipple or to close both eyes.

Facial paralysis is frequently confounded with the facial paralysis of hemiplegia. The patient is believed to have "a partial stroke!" In facial paralysis of cerebral origin the reaction of degeneration is absent, and only the lower facial muscles are affected. The patient can close his eyes as readily as in health, and the emotional movements of the affected side are, in part, preserved.

Paralysis of the nuclei of the facial nerve may produce a paralysis indistinguishable from that due to lesion of the nerve itself. In such cases there is usually associated symptoms referred to other cranial nerves or to involvement of the motor and sensory tracts in the brain.

**Prognosis.**—The prognosis is favorable, so far as partial recovery



is concerned. All cases improve somewhat. The exceptions are those in which the nerve has been divided during surgical operations. Very few cases recover so completely as to leave absolutely no trace of the trouble on careful examination. The majority of cases make what the patient regards as a practically complete recovery. The reaction of degeneration affords an important prognostic guide. As faradic contractility is lost early or late, so will the recovery be prompt or tedious.

**Treatment.**—The first element in the treatment of facial palsy is rest for the affected muscles and the application or retention of heat. Both of these indications may be met by the application of absorbent cotton to the paralyzed parts. Under no circumstances should repair be interfered with by injudicious applications of massage or electricity. Even the investigation for the reaction of degeneration must be conducted with good judgment. After the first one or two weeks, electricity becomes a valuable therapeutic adjuvant. The galvanic current employed should be just strong enough to produce muscular contractions, and no stronger. The sittings should be daily at first, and of about five minutes' duration. The positive electrode should be placed over the point of emergence of the nerve, while the negative should be applied over the motor nerve points of the various muscles in turn and the current interrupted.

The wide-open eye is nearly always a source of great inconvenience, if not actual danger, and may require special treatment. Then the eyelids should be closed and retained in that position by strips of adhesive plaster. Others have recommended that the lids be closed and covered with a shade or bandage; but the pressure required to keep the eye closed under such circumstances is so great as to be very uncomfortable. When the eyelashes are long and firm the lids may be kept closed by hair sutures. In some cases, in which all the above measures have failed to give relief, it may be necessary to stitch the eyelids together.

The sagging of the lower lid produces epiphora, for the relief of which the slitting of the canaliculus is often required.

As to remedies, *aconite* is probably the best in the very beginning, especially in cases arising from exposure to strong cold winds.

*Rhus tox.* comes in when the paralysis is an established fact, especially in those cases arising in individuals of a rheumatic diathesis, as a result of exposure to damp winds. Trousseau and Phillips in their works, praise it highly in various forms of paralysis.

*Causticum*, when the right side of the face is affected. It is useful when the facial palsy is associated with muscular twitchings, or with contractures of the affected muscles.

*Belladonna*, like causticum, will come into play when the right side of the face is affected. In addition to the facial paralysis, there is a neuralgia of the fifth pair of nerves.

*Hypericum* is a possible remedy in those cases where the paralysis has resulted from traumatism of the nerve.

*Gelsemium* is indicated in those rare cases in which facial palsy has followed one of the acute diseases, as diphtheria. It is more frequently indicated in cases with facial neuralgia, and twitchings of the muscles supplied by the seventh pair of nerves.

*Kali hydriodicum* will prove beneficial in cases having a syphilitic origin.

In paralysis depending upon middle ear disease, *silicea*, *hepar*, *mercurius*, *tellurium*, and *aurum*.

If caused by pressure of tumors of the parotid gland, malignant or otherwise, or by pressure of enlarged lymphatic glands, *conium*, *hydrastis*, *baryta iod.*, *calcareo carb.*, *calcareo iod.*, *iodine*, *sulphur*, *arsenic*, *iodide of arsenic*, and *graphites*.

If by periostitis affecting the aqueductus Fallopii, *aurum*, *asafetida*, *silicea*, *mezereum*, *fluoric acid*, *ruta*, and *rhododendron*.

## AFFECTIONS OF THE AUDITORY NERVE.

Our clinical knowledge respecting abnormal conditions of the auditory nerve is almost *nil*. From pathological investigations we are fully aware that this nerve is liable to all the accidents which may befall others. But our means for the recognition of these abnormalities during life are faulty. This lack of practical knowledge would seem to contraindicate a consideration of the subject in a practical text-book like the present one: For myself, I feel that this is a strong reason for treating the auditory nerve difficulties with unusual fulness. The remedy for financial bankruptcy is found in an acknowledgment of the condition and the proper understanding of the causes. The first step to knowledge is an admission of our ignorance.

I have said that the nervous auditory apparatus is liable to all the pathological conditions found in other special sense organs. Thus there may be lesions in the cortical auditory centres (the first temporal gyri), in the nucleus of the nerve about the floor of the fourth ventricle, in the nerve trunk in its course at the base of the brain, and within the bony canal, and lastly in the terminal filaments of the nerve in the internal ear.

The functional disorders may take the form of diminution or deafness, increase or hyperæsthesia or hyperacusis, painful hearing or dysacusis, and perverted function or auditory hallucinations and tinnitus aurium. Under the heading of affections of the auditory nervous apparatus may also be included the consideration of that complex of symptoms generically known as Menière's disease.

**NERVOUS DEAFNESS.** Unilateral deafness from cortical disease is unknown. If the first temporal gyri on both sides are destroyed, complete deafness ensues. Lesions of the first temporal gyrus on the left side produces word-deafness, a condition in which the patient hears sounds but does not understand their meaning. Cases of deafness from a lesion between the cortical centres and the auditory nuclei have been reported as occurring from tumor of the corpora quadrigemina and disease of the posterior part of the internal capsule. In the latter case, the deafness was associated with hemianæsthesia.

The possibility of lesions of the auditory nerve in its intracranial course must be admitted. It is said that an *auditory neuritis* may occur in conjunction with tumors of the brain by the same mechanism that gives rise to optic neuritis. Inasmuch as the latter condition appears not infrequently in these cases without giving rise to local symptoms of any



kind, the same may be true of an auditory neuritis. It therefore becomes a difficult matter to say what means shall be adopted for its recognition. The clinical value of an auditory neuritis is therefore *nil*. Even in cases in which cerebral symptoms are associated with double optic neuritis and disturbances of hearing, the existence of auditory neuritis as the cause of the ear disturbance can only be conjectural. As additional evidence of brain tumor, it must in the present state of our knowledge be regarded as valueless. Pooley reports a case in which the symptoms of Menière's disease were associated with double optic neuritis. The autopsy gave no intracranial solution of the difficulty, and an examination of the internal ear was not made.

A *rheumatic neuritis (rheumatic acusticus paralysis)* similar in character to that involving the facial nerve has been mentioned by Hirt, but its existence has been denied by Gowers.

Disease at the base of the brain may damage the auditory nerve. Thus deafness has been produced by cerebro-spinal meningitis, syphilitic meningitis, various cerebral tumors, and disease of the cranial bones. The principal pathological changes in deafness accompanying the first named of these, are to be found in the labyrinth, and generally the result of a neuritis descendens. The advent of the deafness is associated with vertigo and disorders of gait. The prognosis in these cases is invariably hopeless if the deafness has persisted three months after recovery from the primary disease. In some of these cases the patient can hear certain noises as the cracking of a whip when the human voice is not perceptible.

Primary lesion of the nerve itself is rare. Nerve deafness occurring during the course of locomotor ataxia has been reported and attributed to nerve atrophy, apparently, however, by analogous reasoning from the not infrequent involvement of the optic nerve by that morbid process, rather than as the result of any pathological investigations. Indeed, Lucæ in his examinations failed to find any evidence of nerve degeneration. Buzzard has met with several cases of nerve deafness in association with locomotor ataxia, and quotes Ormerod, who found that condition present in five out of thirteen cases. Buzzard makes the pertinent suggestion that the knee-jerk should be tested in every case of nerve deafness. At present I am treating a man well known in musical circles, and whose case is of great interest in this connection. When first referred to me he suffered from diplopia with absent knee-jerks. He gave a syphilitic history. In the course of treatment ear symptoms appeared, taking the shape of dulness of hearing in one ear, and what he called double hearing. When leading his orchestra, he noticed that certain notes sounded as if a half tone lower by one ear than the other. While this has entirely disappeared and his diplopia is nearly well, he has other symptoms which point to a possible development of other symptoms of locomotor ataxia.

The terminal filaments within the internal ear may likewise be affected by acute or chronic inflammation, syphilitic disease, and degenerative processes.

Functional nerve deafness is a possibility, but not much more than that. It has been alleged to occur as an hysterical symptom, and as one of the symptoms of lithæmia. Regarding the former, the relationship is not always clear. Severe aural symptoms, as deafness, vertigo, and tinnitus, may readily produce marked hysterical manifestations in the predisposed. Again we are all aware that hysterical patients may simulate very closely almost every possible phase of organic disease. As to lithæmia, it may well be questioned if the deafness sometimes accompanying that condition is not the result of one of the numerous pathological changes to which that variety of toxæmia may give rise.

Constant overtaxation of the hearing may produce a very peculiar general nervousness, and even quite serious effects on the mind.

Defects in hearing dependent upon depressing causes, neurasthenia, sexual excesses, overwork, are not necessarily of nervous origin, as they may very readily occur from alterations in the vascular supply of the middle ear and the Eustachian tubes.

Nerve deafness has been known to follow typhoid fever, scarlatina, smallpox and mumps.

The differentiation of the various auditory nerve lesions is practically impossible. To distinguish nerve from middle ear deafness is not so difficult. The ability to hear better and longer the sound from a tuning-fork, or other test object, when held some inches from the ear in front of the auditory meatus, than when placed against the mastoid in actual contact, is indicative of disease of the nervous apparatus.

Labyrinthine and nerve disease can only be differentiated by the associated symptoms. In the former, deafness, tinnitus and vertigo constitute the usual symptomatic totality. In the latter there will not infrequently be evidence of implication of other nerves and of cerebral structures.

The auditory nucleus may be affected by hæmorrhage, softening or degeneration. Deafness from this cause may be suspected when it appears suddenly. Degeneration of the auditory nuclei only occurs in association with extensive bulbar disease.

*Hyperæsthesia* of the sense of hearing may occur in hysteria and at the onset of certain acute cerebral and general disorders. A form of hyperæsthesia, in which the patient is able to discern sounds not perceptible to the healthy, has been described.

*Dysæsthesia*, in which sounds cause discomfort, may occur in quite a variety of diseases, but possesses no diagnostic significance.

*Toxic deafness* is undoubtedly of nervous origin. The two drugs which are pre-eminently able to give rise to it are quinine and the salicyl-

ates. The knowledge of this fact is very important, in view of the very frequent abuse of both drugs in the treatment of malaria and rheumatism respectively. The deafness may be permanent. Its onset is often characterized by vertigo and tinnitus. It is interesting to note that both remedies are highly praised by old-school authorities in the treatment of aural vertigo.

Abnormal sounds partake of two characters, namely, tinnitus aurium and auditory hallucinations.

*Tinnitus aurium.* This is generally understood as a subjective sense of ringing in the ears or head. The character of the subjective noise, however, presents the widest variations. It may be hissing, sizzling, clanging, hammering, etc. It is more or less constant, and of variable duration. When of mild degree, it may be noticed only when outward quiet reigns. Sometimes the sounds are of a highly specialized character, and more or less paroxysmal. The pathological causes of tinnitus are found most frequently in the middle ear, organic lesions which irritate the auditory nerve, and certain blood changes, as anæmia, lithæmia, etc.

*Auditory hallucinations* are not infrequent in insanity and quite a variety of organic intracranial conditions, as hæmorrhagic pachymeningitis, atrophy of the brain, external hydrocephalus, etc., tumors in or adjacent to auditory centres. They have been known to constitute the aura in cases of epilepsy, and they have occurred from labyrinthine changes. Subjective sounds starting as tinnitus have been converted into hallucinations.

### AUDITORY NERVE VERTIGO.

Vertigo is a not infrequent symptom attendant upon aural disease. It may arise in conjunction with disorders of the external meatus and middle and internal ear. As a symptom of lesion of the meatus it is found especially in association with foreign bodies, as impacted cerumen. I can recall one very aggravated case illustrating this very point. The patient had been refused insurance because of a persistent vertigo from which he had suffered for some time. Examination of his ear revealed the presence of a large and very hard plug of cerumen. This was at once removed with the forceps, its withdrawal causing the patient to faint. The vertigo then disappeared only to return some time afterwards, when the meatus again became clogged with hardened wax. The slightest manipulation of this man's ears always gave rise to vertigo; even the gentlest syringing which was employed on subsequent occasions necessitated his lying down from time to time.

While the presence of foreign bodies in the auditory meatus may thus produce discomfort, it is singular that boils and inflammatory conditions of this part generally do not excite any vertiginous sensations as part of their symptomatologies.



Middle ear disease is sometimes accompanied by vertigo, generally through pressure exerted upon the oval window. Vertigo from this cause is usually promptly relieved by Politzerization.

Vertigo from internal ear disease occurs only when the nerve filaments within the semicircular canals are irritated or compressed. Respect for a correct nomenclature should lead to this form of vertigo being styled auditory nerve vertigo, but common usage has led to the title "Menière's disease." In 1861, Menière described a number of cases characterized by sudden paroxysms of tinnitus associated with vertigo and followed by deafness. Sometimes the patient became partially unconscious during the seizures, and gave evidence of very profound circulatory disturbance. One of the cases, that of a girl aged nineteen years, who had caught cold during menstruation, died. An autopsy showed no intracranial changes to explain the severe and remarkable symptoms, but the semicircular canals were filled with a bloody serum.

Literature since the discovery of this disease has made the subject very confusing, because it is evident that authors describe very different pathological conditions under this name. The title "Menière's disease" should be abolished, and if the name of this physician is to be perpetuated in medical nomenclature, that of "Menière's complex of symptoms" substituted. It is plain that these symptoms may arise in connection with disease of the semicircular canals from almost any pathological change.

The prognosis of auditory nerve vertigo is, as a rule, favorable. Still a number of cases end fatally, and still more go on to complete deafness, at which time the vertigo disappears.

## VERTIGO.

While on this subject, I feel that it is well to refer to that of vertigo in general, especially as investigators have of late years been inclined to attribute many of the vertigos formerly assigned to disease of various viscera, as having some relation to the hearing apparatus. Arising from whatever cause it may, vertigo presents essentially the same features, the recognition of its origin depending upon a study of the associated symptoms. It may arise from (1) organic disease of the central nervous system, (2) disease of the circulatory apparatus, (3) epilepsy, (4) hysteria, (5) peripheral irritation, (6) disease of the special senses, (7) toxæmia, and (8) from conditions which in the present state of medical knowledge are undiscoverable; this class of cases being generally spoken of as essential vertigos.

Taking up these varieties of vertigo *seriatim*, we come first to *organic vertigo*. This may accompany disease of any character situated in any portion of the brain. It is, however, especially liable to occur in connection with tumors, particularly so when they are situated in or near the

cerebellum. Still it is perfectly possible, indeed not infrequent, for tumors and lesions of the cerebellum to occur without the slightest disturbance in equilibrium. Vertigo is a not infrequent accompaniment of locomotor ataxia and disseminated sclerosis of the brain and spinal cord.

*Vertigo from cardiac disease* is the result of failing circulation. It is, therefore, aggravated by exertion, strong emotions, and causes calling upon the heart for extra work. It is a symptom of fatty degeneration and valvular disease of the heart. Occasionally it occurs from venous plethora of the brain.

Vertigo sometimes occurs in the form of *epileptic* seizures. Under such circumstances the attack is ushered in without warning, as in Menière's complex of symptoms, and may be associated with deafness and tinnitus. Epileptic vertigo is readily differentiated from the other by the very quick recovery of the patient, the loss of consciousness and the absence of deafness when the attack subsides, which is usually within one or two minutes. If these data are insufficient, one may fall back on the history of the case, which will tell of other attacks and of convulsive seizures.

*Hysterical vertigo* is recognized by its remarkable features, unexplainable on any other hypothesis. It is, however, very rare.

Vertigo occurs very frequently in association with *neurasthenia*. It is then dependent upon the readiness with which the hyperæsthetic nervous system responds to peripheral irritation. In other cases it is the result of cerebral anæmia.

Of *peripheral vertigos* we have two that have obtained especial prominence; these are gastric and laryngeal. The former of these is not as common as was at one time supposed. Still vertigo does occur with a fair degree of frequency in connection with acute indigestion. In chronic gastric disturbances, it is not so often observed. In the latter cases, it may be the result of a toxæmic condition produced by the absorption of imperfectly digested food products. Gastric vertigo may be associated with vomiting, headache, or obscuration of vision.

*Laryngeal vertigo* was first described by Charcot in 1876. It begins with a sensation in the larynx, which leads to a severe, spasmodic cough, at once followed by vertigo and unconsciousness. The attacks last for but a few minutes. Physical examination shows quite a variety of conditions present in these cases. Almost any anatomical condition may be present; and again, the larynx may present all the appearances of perfect health. By many this variety is regarded as of epileptic origin.

Vertigo may be a prominent symptom in cases of *ocular palsy*. It does not seem to be caused by the diplopia, for it still persists when the normal eye is closed. It appears to be the result of the erroneous projection of the visual field by the paralyzed eye. One quite often experiences vertiginous sensations when visual perceptions are at variance

with perceptions obtained simultaneously through other senses. Within a short time, this fact was very forcibly illustrated in my own person. While riding backwards in a railroad-car, the train stopped at a station. A freight train shortly came up, when the train, on which I was, started. The sensation transferred to my body told me that I was going backwards, but looking at the freight train which was moving faster than my train, my sight told me that I was moving forward. As soon as the speeds of the two trains became equal, and when my train travelled faster than the other, the vertigo disappeared.

The vertigo from seasickness and swinging, etc., is due to disturbance of the circulation within the semicircular canals. It is a very interesting fact that deaf people are rarely affected with seasickness.

The *toxæmic* conditions which produce vertigo, are quite varied. Quite a number of poisonous drugs may produce it, *e. g.*, belladonna, cannabis indica, alcohol, etc. Lithæmia may give rise to very severe vertigo. The uræmic manifestations of Bright's disease include vertigo. It has seemed to me, however, that when this symptom is associated with chronic renal disease, it very often depended upon the increased arterial tension and the cardiac hypertrophy.

**Gerlier's Disease; Paralyzing Vertigo.**—This disease was first described by Gerlier. It exists only in a very limited locality in Switzerland and southern France. Its characteristic symptoms are vertigo, cervico-occipital pain, and certain palsies. These occur in paroxysms, which are usually of short duration, that is, of not more than ten minutes. The paralyzes are observed in the upper lid (ptosis), and in the extremities. The pathology of Gerlier's disease is obscure. By many it is attributed to a specific microbe developed in the stables during the summer months; by others, the contagion of fear in a locality, predisposed by superstition, is regarded as an important factor. With the advent of cold weather, the disease disappears.

**Treatment.**—The treatment of vertigo must vary according to the cause. No extended consideration need, therefore, be given here. The reader is referred to the various chapters treating of the diseases causative of this symptom.

The following therapeutic suggestions are offered :

For auditory nerve vertigo, *salicylate of soda*, *quinine*, *gelsemium*, *causticum* and *bisulphide of carbon*. Salicylate of soda and quinine should be used in rather crude doses, that is from one or two grains of the first decimal trituration to three grains of the crude drug three or four times daily. The *salicylate* is probably the better, and is the one with which I have obtained the most satisfactory results. *Gelsemium* is highly recommended by Ringer, who prescribes ten drop doses of the tincture three times daily.

*Causticum* is useful not only in auditory vertigo, but in other varie-



ties as well. It finds its main use, however, in the vertigos of organic brain disease; the disordered equilibrium is associated with mental weakness and constant feeling of anxiety.

For the vertigo of gastric disease no remedy equals *nux vomica*.

*Conium* is the remedy for the vertigo of the aged, and for the variety resulting from sexual excesses, especially from masturbation.

*Plumbum*, *argentum nitricum* and *anacardium* are all remedies for the vertigo of organic cerebral affections.

For vertigo of heart disease, *digitalis*.

For that of renal disease and general arterio-sclerosis, *glonoin* in the first centesimal dilution, one drop from three to five times daily.

For purely nervous vertigos, *theridion*, *ambra grisea*, *sanguinaria* and *phosphorus*.

For congestive vertigos, *belladonna*, *glonoin* and *stramonium*.

For anæmic vertigo, *cinchona* and *ferrum*. The latter drug had best be administered in the form of iron reduced by hydrogen, from one to two grains three times daily.

## AFFECTIONS OF THE GLOSSO-PHARYNGEAL NERVES.

Our knowledge respecting the functions of the glosso-pharyngeal nerve is highly unsatisfactory. This is due to its anatomical peculiarities, the nuclear cells from which it arises being closely associated with those of the pneumogastric and spinal accessory nerves, and to its extensive communications with the fifth and seventh pairs of nerves. Pathological study has not been of any help in extending our knowledge, for this nerve or its nuclei are practically never involved alone. We have very good reason to believe its functions are of a mixed character. The nuclear cells from which it arises are large, and of the character subserving motor functions. It is believed to be the motor nerve of the stylo-pharyngeus muscle and the middle constrictor of the pharynx. In spasm dependent upon disease of this nerve we have, therefore, spasmodic dysphagia. Paralysis gives rise to difficulty in swallowing, and occurs as a symptom of brain tumor, meningitis, and post-diphtheric paralysis. It must be remembered, however, that it is very rarely that the glosso-pharyngeal nerve is affected alone.

It is believed, too, that the glosso-pharyngeal is the nerve of common sensation for the upper part of the pharynx and back of the tongue. Some authorities deny this, however.

The relation of the glosso-pharyngeal nerve to the sense of taste is just as unsettled as are its other functions. Most authorities hold to the opinion that it is the special nerve of taste for the back of the tongue, the fifth pair supplying the anterior half of that organ. Hirt assumes that it is "the only genuine nerve of taste." Gowers says "that there is no instance on record of loss of taste at the back of the tongue from disease of the roots of the glosso-pharyngeal nerve, while there is evidence that disease of the roots of the fifth nerve causes loss of taste on the back as well as on the front of the tongue, and also on the palate and palatine arch." Dana says cases in which paralysis of the glosso-pharyngeal has caused complete unilateral loss of taste have been reported. The inevitable conclusion from these statements is that those fibres of the glosso-pharyngeal subserving taste come ultimately from the trigeminus. Putting aside all physiological discussions, a few words concerning disease of the sense of taste are in order.

**Ageusia.**—By this is meant a diminution or loss of the sense of taste. In testing the sense of taste, we test only for sweet, sour, salty and bitter. This may be done by applying to the tongue in turn sugar,

vinegar, salt, and quinine. The tongue is to be protruded. The test substance is applied first to the back of the tongue on one side and then on the opposite. Next the anterior half of the tongue is tested in the same manner. Care must be taken, however, that the patient does not draw his tongue back into the mouth in the course of the experiment. The test for "bitter" (quinine) should be applied last, as otherwise it will interfere with the taste of the other substances.

Ageusia is observed very frequently in connection with facial palsy, the anterior half of the tongue being affected. It may also occur in hysteria and in injuries to the trigeminal and glosso-pharyngeal nerves. It may occur independently of nerve disease from the dry mouth of fevers, and in catarrhal inflammation of the mucous membrane of the mouth. It has been reported as of bulbar origin, then being due to nuclear degeneration, especially in association with tabes dorsalis.

**Parageusia** is perversion of the sense of taste. It may occur in gastric disorders, and in hysteria and insanity.



## AFFECTIONS OF THE PNEUMOGASTRIC NERVES.

The extensive distribution of the pneumogastric nerves, sending as they do branches to the pharynx, larynx, œsophagus, trachea, bronchi, lungs, heart, stomach, intestines and spleen, exposes them to not infrequent lesions. A description of disease as it affects them is rendered somewhat difficult by reason of their close anatomical connection with the glosso-pharyngeal and spinal accessory nerves, from the latter of which they derive their motor fibres to the larynx. The pneumogastric nerves may be affected by reason of disease in their nuclei or in the course of the nerve trunks themselves. Nuclear degeneration rarely occurs as an independent affection, being usually a part of the widespread bulbar changes constituting the anatomical substratum of glosso-labio-laryngeal paralysis, or of the degenerative tendency manifested in disseminated sclerosis, general paralysis of the insane, and locomotor ataxia. The roots of the nerves are sometimes affected by lesions of contiguous structures, notably by thickening or inflammation of the meninges, new growths, and aneurisms of the vertebral artery. Syphilis is often found to be at the bottom of the trouble under these circumstances. The trunk of the nerve may be diseased or injured in any portion of its course. It is especially liable to injury from gunshot and stab wounds, aneurisms, and from tumors of the neck and thorax. Like all other nerves it is liable to inflammation, as the result of cold or toxic influences. Pneumogastric neuritis is occasionally observed following diphtheria, and is probably the cause of quite a number of sudden deaths during convalescence. We now proceed to the study of the lesions of the branches of distribution.

**Pharyngeal Branches.**—The plexus of nerves distributed to the pharynx is made up of filaments obtained from the glosso-pharyngeal and pneumogastric nerves. As to the relative importance of the fibres of the component nerves in subserving the motor and sensory functions of the part, and their areas of distribution, absolutely nothing is known. We do know, however, that pharyngeal paralysis results from nuclear disease in the majority of cases, and is a frequent symptom in post-diphtheritic paralysis.

Some few cases also arise as the result of meningeal disease, and lesions of the bones, but such cases are necessarily very rare. Pharyngeal paralysis from lesion of the nerve itself outside of the skull is probably unknown.

**Symptoms.**—Dysphagia is the chief symptom of pharyngeal paralysis. The bolus of food lodges in the pharynx, instead of being forced onwards into the œsophagus. Liquids are often ejected through the nose, due probably to non-involvement of the middle constrictor of the pharynx. If the lesion is unilateral, the difficulty in deglutition is very slight.

Pharyngeal paralysis may be confounded with malignant disease of the œsophagus, the sole ground for error being the presence of difficulty of swallowing which is found in both affections. The differentiation of the troubles is readily made by the use of the œsophageal sound, which passes readily in the case of paralysis.

*Pharyngeal spasm* is met with almost exclusively in hysteria, and is the cause of the *globus hystericus*.

**Laryngeal Branches.**—The study of laryngeal palsies is a most interesting one. The branches going to the muscles of the larynx are two in number, viz., the superior laryngeal, which supplies the crico-thyroid muscle and the epiglottis, and the recurrent laryngeal, which furnishes motor power to all the other muscles of the larynx. According as one or the other of these nerves or their ultimate branches are diseased, certain groups of objective phenomena are presented. The most frequent causes of laryngeal palsies are found in degeneration of the nuclei of the spinal accessory, and damage to the recurrent laryngeal from pressure as from aortic aneurism. Very exceptionally disease of the cortical centre for the larynx may cause destruction of the functions of that organ. To do so, however, it is necessary, that the cerebral disease be bilateral. Not uncommonly one meets with functional aphonia in hysterical and anæmic females.

The paralysis may be widespread, that is, involve all the laryngeal muscles supplied by one nerve, or it may be limited strictly to one or two muscles. In the latter case, the paralysis producing lesion is local, *i.e.*, laryngeal, arising from nerve degeneration secondary to catarrhal or other inflammations.

The objective symptoms on which we rely for the differentiation of the different forms of laryngeal palsy are those referred to the voice and respiration, and those obtained by laryngoscopic observations. For correctness of observation with the laryngoscope, it is necessary to understand the positions of the vocal cords at absolute rest, and during phonation and respiration. When at absolute rest, the vocal cords assume a position of slight abduction from the median line. During phonation they are approximated and made tense. During inspiration they separate, the degree of separation depending upon the force of the inspiratory act. During expiration they approach each other slightly.

*Complete paralysis of all the muscles supplied by the recurrent laryngeal nerves.* The vocal cords are observed to stand in the position of complete

equilibrium, the cadaveric position so-called. There is complete loss of voice, and the cords do not move during either respiration or attempted phonation. There may be slight stridor. The power to make an explosive cough is lost. In unilateral paralysis of the recurrent laryngeal, one cord is observed to be entirely motionless, while the other cord moves normally during phonation and respiration. The voice is low pitched and hoarse. Stridor is absent. When the lesion is such as to but partially paralyze the muscles supplied by the recurrent laryngeal, the result is an apparent abductor paralysis.

*Paralysis of the abductors.* The cords are seen to be near together and do not separate during inspiration. The voice is but little, if at all, affected. The respiratory symptoms are both remarkable and characteristic. The failure of the cords to separate during inspiration leads to a whistling stridor during that act, while expiration is unimpeded. Explosive coughing is unimpaired. This condition involves considerable danger, as a very slight swelling of the mucous membrane covering the cords may lead to death from asphyxia. Abductor paralysis occasionally comes on as the result of cold or laryngeal catarrh. Unilateral abductor paralysis produces very little if any effect on either voice or respiration. Its discovery rests upon laryngoscopic examination. An incomplete lesion of the recurrent laryngeal nerves may lead to a close simulation of the objective equivalents of abductor paralysis.

Abductor paralysis occasionally occurs as a manifestation of hysteria, and is then accompanied by the usual symptoms of this condition, inspiratory stridor with normal voice. These cases offer a favorable prognosis, although it is said that occasionally a fatal result ensues. Gowers suggests that many of the cases of so-called hysterical spasm of the glottis are really examples of hysterical abductor palsy.

*Paralysis of the adductors.* In this the vocal cords stand apart and are not approximated during attempted phonation. They separate still further during inspiration. They can be brought together during coughing. This variety of laryngeal palsy is very common as a manifestation of hysteria.

This form of paralysis may result from local causes, especially from excessive use of the voice, and laryngeal catarrh. According to Bose, it may follow cold without the intervention of laryngitis. In some of the hysterical cases, the patient is able to sing or speak under the influence of a powerful emotion, when ordinary conversation is impossible excepting in a whisper.

The following table, modified from Hirt's work on Nervous Diseases, epitomizes the diagnosis of laryngeal palsies:



KIND OF PARALYSIS.	OCCURRENCE.	SYMPTOMS.	LARYNGOSCOPIC PICTURE.
Complete recurrent palsy.	In compression paralysis of the vagus or the recurrent laryngeal (carcinoma œsophagi), often unilateral (left), as initial symptom of aortic aneurism. In tabes.	Voice not clear; patient is easily tired on talking; coughing impossible.	Vocal cords slightly abducted, the so-called "cadaveric position." In forcible phonation, the healthy cord reaches beyond the middle line. Over-riding of the arytenoid cartilages.
Abductor paralysis (paralysis of the post-crico-arytenoids).	In diseases of the nerve itself, the causes of which are often unknown.	If bilateral: extreme inspiratory dyspnoea; if unilateral: inspiration hampered, long-drawn noisy. Dyspnoea on the least exertion. Speech but little affected.	Glottis appears as a narrow slit, becoming still narrower on inspiration. Inability to abduct the paralyzed vocal cord.
Paralysis of the internal thyro-arytenoids.	In catarrhs of the mucous membrane of the larynx. After over-exertion of the voice. In hysteria.	Voice hoarse; speaking an effort.	Glottis does not close completely on phonation. If at the same time the arytenoids are paralyzed, the glottis presents an hour-glass outline. Neither anterior nor posterior position is closed, but the vocal processes are in their normal position.
Abductor paralysis.	Rarely isolated. In hysteria.	Absolute absence of voice. Power of coughing retained. "Phonic paralysis" (Turck).	Cords normal in position, moving normally during inspiration, but not approximated on phonation.
Paralysis of the crico-thyroids.	After diphtheria.	Voice rough; high tones impossible.	Excavation of the vocal cords. Cords do not vibrate visibly.

**Sensory Disturbances of the Larynx.**—These consist of anæsthesia and hyperæsthesia. **ANÆSTHESIA** appears usually unassociated with motor palsy. It is generally limited to the area supplied by the superior laryngeal nerves, and is a prominent symptom in some cases of post-diphtheritic paralysis. The symptoms of laryngeal anæsthesia are not very obtrusive. The condition is recognized by physical exploration,

the manipulations of the throat and larynx incidental to laryngoscopic examination fail to excite reflex gagging and coughing. There is liability of food to enter the larynx, because of failure of the epiglottis to close promptly. This accident may have a disastrous result, as it is not unlikely to produce deglutition pneumonia.

Laryngeal anæsthesia may be of hysterical origin, in which case it sometimes forms part of a general hysterical hemianæsthesia. Touching the mucous membrane of the larynx in these cases excites reflex coughing as in health, thus differentiating the anæsthesia from that due to nuclear degeneration.

HYPERÆSTHESIA OF THE LARYNX is very rare as an independent condition. It is usually symptomatic of ulceration, or other local disease.

LARYNGEAL SPASM. In this the muscles closing the larynx, the adductors and the muscles of the epiglottis, are involved. It takes very little local irritation in health to excite a reflex response from these muscles. In case of local disease, catarrhal inflammation, or ulceration, more or less laryngeal spasm is to be expected. The attacks are always worse at night, and are always bilateral.

The most common form of laryngeal spasm is that known as *laryngismus stridulus*. It occurs in children mostly, especially in those of rachitic constitution. It depends upon a morbid reflex excitability. There are no local changes. The attacks are liable to occur from the slightest peripheral impression, or even without apparent cause. They are sometimes observed in tetany. Among the laity they are referred to as "inward spasms." Owing to the old hypothesis that the disease was associated with enlargement of the thymus gland, it has been called "thymic asthma." The attacks may come on at any time in the twenty-four hours. The child is unable to get its breath, struggles and becomes cyanotic. The spasm then suddenly relaxes with a high-pitched crowing sound.

Attacks of laryngeal spasm, similar to the bronchial spasm of asthma, have been observed in adults. They sometimes replace attacks of migraine, and are symptomatic in locomotor ataxia, and some few central affections. Laryngeal spasm is of occasional occurrence in neurotic troubles, and doubtless explains the cyanosis sometimes observed in hysterical females.

There is a rare form of spasm described by Gowers, in which the morbid movements are excited by attempts to speak. I have seen it in adults, wherein even the slightest coughing-spell would terminate in an adductor spasm.

**Œsophageal Branches.**—These are practically never paralyzed excepting in nuclear disease. Œsophageal spasm is of occasional occurrence in hysteria. It is called œsophagismus. It sometimes occurs as a reflex phenomenon, as a result of indigestion, protracted vomiting, and

faucial irritation. Symptomatically, it is recognized by inability to swallow, and regurgitation of the food after it reaches a certain spot. It is differentiated from organic stricture of the œsophagus by the history of the case, and by the fact that in the spasmodic variety the patient is sometimes able to swallow without difficulty. In some of the cases the spasm gives rise to so much pain as to lead to entire refusal of food, and consequent emaciation. It must not be forgotten, however, that an apparently spasmodic œsophageal stricture may have an organic stricture of lesser degree as its exciting cause.

**Gastric Branches.**—The pneumogastric nerves are the principal sensory nerves of the stomach, and furnish part of the motor supply to that organ. Disturbance of these nerves therefore may give rise to neuralgia and various sensory and motor disturbances of the stomach. These will be considered fully under the titles of gastralgia, gastric neuroses, and nervous dyspepsia, in another section of this work. I will dismiss the consideration of the gastric symptoms dependent upon pneumogastric trouble by mere mention of the gastric crises observed in some cases of ataxia, and reflex and other forms of nervous vomiting.

**Cardiac Branches.**—Disease of these may produce quite a variety of cardiac disturbances, notably angina pectoris, palpitation, rhythmical disturbance, and various morbid sensations referred to the cardiac region.

**Pulmonary Branches.**—Conditions dependent upon disease of these are not well understood. Asthma is now regarded as a pneumogastric neurosis, the consideration of which will be deferred to another section of this work.

**Prognosis.**—The prognosis must depend upon the nature of the primary trouble. Pharyngeal and laryngeal paralyses of diphtheritic origin generally make a most excellent recovery, although when severe they involve considerable danger to life. Paralyses dependent upon nuclear degeneration are incurable. Those arising from pressure on the nerve roots by syphilitic exudation, if taken early, will probably make a good recovery. Functional cases offer a favorable prognosis, though they oftentimes resist well-conducted moral and medical treatment for a long time. Paralysis of the abductors of the vocal cords is attended with considerable danger to life, owing to the liability of complete occlusion of the lumen of the larynx. Laryngismus stridulus offers a favorable prognosis in the majority of cases.

**Treatment.**—In pharyngeal and laryngeal palsies electricity is the most important element in the treatment. Faradism or galvanism may be employed. When the pharynx is the part to be stimulated, one internal and one external electrode should be used. The first-named (the negative) should be in the form of a swab well saturated with salt water. The latter should be an ordinary flat electrode. In the case of



laryngeal palsy the application may be made by a bipolar electrode, one internal and one external electrode, or both electrodes external. The two former methods require considerable manual dexterity and technical skill for their successful performance, and, owing to their painfulness, cannot be prolonged more than a few seconds. The last-named method is as efficient as the others, often more so, for by it the dosage can be made much greater. In functional palsies one method often succeeds when the others fail. The internal applications are the more efficient. Still I have seen a most remarkable cure of a functional aphonia after one external application of faradism. Sometimes static electricity is especially successful in functional palsies. The best method is the withdrawal of sparks from the larynx.

In cases of abductor palsy the greatest care is needed in electrical applications, lest the stimulation be wrongly applied, and the adduction increased. Death from asphyxia may thus be caused. It is oftentimes necessary in these cases of abductor paralysis to perform tracheotomy and have the patient wear the tracheotomy tube permanently.

Functional aphonias sometimes make most remarkable recoveries after nervous shocks, as from fright, excessive joy, and other emotional influences. Sometimes good moral management of the physician is required. I once cured a very obstinate case, that had resisted electrical and other treatment, by anæsthetizing the patient and keeping her in the first stage of anæsthesia for several minutes. During this time she began to talk freely in her normal voice. No relapse has occurred to date.

Massage of the larynx may be advised in some cases.

Rest of the voice is an important desideratum in cases of diphtheritic and other palsies dependent upon neuritis. In the early stages of these forms of paralysis *gelsemium* and *aconite* are the preferable remedies. In the majority of cases the former will be found to be the indicated remedy.

*Rhus* and *causticum* are indicated in the rheumatic cases. The former is to be selected on its general indications; the latter by the local paralytic phenomena. *Causticum* is one of the few remedies that may possibly do good in nuclear degeneration.

*Phosphorus* is unquestionably the best remedy for paralytic aphonias secondary to laryngeal catarrh. Next to it in value is *rumex*.

Functional aphonia calls for *ignatia*, *cocculus*, *nux moschata*, *phosphorus* or *sepia*.

*Laryngismus stridulus* requires a thorough examination of the patient to insure the removal of all possible sources of irritation. Ringer has recommended that in severe cases the child be placed in a bath of warm water two or three times daily, while the back and chest are thoroughly sponged with cold water. During the paroxysms themselves, relief is to

be obtained by dashing cold water on the face. The greatest care must be exercised concerning the diet. Many of these cases occur in poorly-fed children. Fresh air and good milk afford the readiest means of cure. The remedies may be selected according to constitutional or local phenomena, generally the former. The remedies from which a selection is to be made are the *calcareas*, *bromine*, *chlorine*, *cuprum*, *gelsemium*, *ignatia*, *iodine*, *ipecac*, *lachesis* and *sambucus*.

## AFFECTIONS OF THE SPINAL ACCESSORY NERVES.

The spinal accessory nerves consist of two portions, one arising from the medulla, and the other from the spinal cord. The former, generally spoken of as the accessory portion of the nerve, joins the pneumogastric, and furnishes motor power to the laryngeal muscles. Disorders of this branch were described in the previous section. The remaining branch is really a spinal nerve which pursues the unusual course of passing into the cranial cavity before proceeding to its points of distribution, namely, the sterno-mastoid and trapezius muscles. The spinal accessory is a purely motor nerve, hence disease of it is characterized by paralysis or spasm, as the case may be. It may be damaged in any portion of its course by injury or disease. Nuclear degeneration of the accessory portion is observed as a part of progressive bulbar paralysis. The nuclear cells of the spinal portion sometimes degenerate in progressive muscular atrophy, and are occasionally involved in poliomyelitis anterior. The nerve fibres themselves may be compressed by meningitic exudations and tumors within the skull, or by enlarged glands, and tumors externally. They are occasionally involved in cranial fractures, and may be damaged in cases of vertebral caries when the disease is high up. A congenital paralysis of this nerve is occasionally encountered, and is believed to be due to injury to the nerves during labor. It usually escapes early detection owing to the short round neck of infants, and the fact that the little ones, even in health, are unable to hold the head erect at first.

**Symptoms.**—Paralysis of the sterno-mastoid and trapezius only will here be considered. According to the seat of the disease, both muscles, or the trapezius alone may be affected. Paralysis of the sterno-mastoid is indicated by the loss of the usual sharp contour of the muscle observed during movement of the head, and by inability of the patient to rotate the head towards the side opposite to that of the paralyzed muscle. There is no deviation of the head when at rest. Later there is generally a secondary contraction of the opposite muscle, in which case a *so-called* paralytic torticollis develops.

Paralysis of the trapezius is never complete from disease of the spinal accessory nerve alone, for that nerve only supplies exclusively the upper portion of the muscle, the remainder receiving its motor power from spinal nerves. This partial paralysis of the trapezius is recognized by the alterations in the contour of the outer side of the neck. Nor-



mally, this is nearly a straight line; with the upper portion of the trapezius paralyzed, it assumes a concave curve, which is made much more prominent by deep inspiration.

Paralysis of the middle portion of the trapezius is indicated by slight drooping of the shoulder, recession of the scapula from the spinal column, and rotation of its inferior angle inwards. Ability to raise the arm is impaired, because the fixation of the point of support for the deltoid is lessened. The middle portion of the trapezius is never completely paralyzed from spinal accessory disease alone, as it receives nerve supply from spinal nerves.

In bilateral sterno-mastoid paralysis the head falls backwards; in bilateral trapezius paralysis, forwards.

**Diagnosis.**—The only difficulty is found in deciding as to the portion of the nerve affected. In intracranial disease other nerves are apt to be involved. Thus we may find involvement of the laryngeal and lingual muscles. If the nerve is injured after passing through the sterno-mastoid, then the trapezius only will be affected.

**The Prognosis** depends entirely upon the nature of the primary disease.

**Treatment.**—No special treatment need be outlined for these cases. Electricity and massage are invaluable when no inflammatory action is taking place. In all cases in which the paralysis is secondary to other diseases, as Pott's disease of the spine, syphilitic meningitis, traumatism, etc., these conditions must be regarded as affording the most important indications. As to remedies, the reader is referred to the sections treating of the primary diseases.

## TORTICOLLIS.

The term torticollis is used to designate a variety of conditions in which an abnormal position of the head in its relation to the trunk is produced by muscular contractions. There is also a form of mal-position dependent upon disease in other than the nervous and muscular structures, generally upon disease of the cervical vertebræ. To this the name "*false torticollis*" has been applied. Its recognition is very important because of the therapeutic indications thereby furnished. We have also the so-called "*rheumatic torticollis*," caused by acute myositis, and this is characterized by pain and tenderness of the muscular substance. "*Congenital torticollis*" is a variety in which the deformity is dependent upon permanent shortening of a muscle, usually the sterno-mastoid. It is believed to be due to an intra-uterine or obstetrical injury of the sterno-mastoid, as it occurs most frequently in presentations. A peculiarity of this variety is its remarkable tendency to association with facial asymmetry. The affected muscle is found to be hard and firm, and the essential muscular elements more or less atrophied. "Spasmodic torti-

collis" is the name given when the deformity depends upon instability of nerve-cells. It is of two varieties, tonic and clonic. In the former, the spasm is persistent and prolonged, assuming the appearance of a contracture. It is limited to one side and usually to one muscle. When the sterno-mastoid is involved, the face is rotated to the opposite side, the chin somewhat elevated, and the head inclined to the affected side. When the trapezius is affected, the inclination of the head is more marked, the head is drawn backwards and the shoulder is raised. Permanent contracture ensues in cases of long standing.

The clonic variety is of varied distribution. It involves one muscle or many; those of one side or both. Beginning in one of the muscles supplied by the spinal accessory nerves, it may extend and be widespread. The spasms may be few in number, occurring at long intervals, or, again, they recur in such rapid succession as to be disabling.

Prominent among the causes of spasmodic torticollis is the neurotic constitution. This is exhibited by the presence of cases of other nervous diseases, as epilepsy, migraine, etc., in different members of the family, and by a personal history of other neuroses in former years. Women seem to be affected with greater frequency than men. The disease is rarely observed prior to middle life. Cases occurring in women of less than thirty years of age are, in all probability, of hysterical origin. Injury of the parts, depressing emotions, otitis media, excessive use of the muscles, and malaria, have been assigned as causes in individual cases. It is very rarely, indeed, that it occurs from peripheral irritation.

The pathology of the disease is not at all understood. In no case has an autopsy revealed a lesion explaining the symptoms. It is believed that the morbid movements depend upon the overaction of nerve-cells. As to the situation of the nerve-cells, we must rely wholly on conjecture. It has been observed that in some cases the muscles affected are those physiologically associated in their functions. In such it is fair to presume that the affected cells are of the higher centres, *i. e.*, the cortical. When no such association is found, the lower centres are the seat of disease. As to the nature of the changes which produce the overaction of the nerve-cells we must express complete ignorance.

**Symptoms.**—Clonic spasms of definite distribution, but varying according to individual cases, comprise nearly the entire symptomatology of spasmodic torticollis. Usually spasm is the first symptom. It appears gradually, first in one muscle, slowly increasing in intensity and spreading to others. Sometimes this spasm is preceded or accompanied by slight sensory disturbance, as pain or sensation of stiffness. It is generally aggravated by emotional influences, if, indeed, it is not present only at such times. The muscles most commonly involved are the sterno-mastoid and trapezius, and, next to these, the splenius capitis. It is an important thing, from a therapeutic standpoint, to determine the muscle

in which the spasm commenced, as sometimes the removal of the spasm from that muscle by surgical measures stops it in all secondarily affected. As the spasm extends, the muscles of the face and arm become involved. The abnormal movements always occasion fatigue and discomfort, but rarely absolute pain. Sometimes numbness and tingling in the arm are associated.

**Diagnosis.**—The most important point is the differentiation from false torticollis. In this condition, the tense muscle is found on the side towards which the head is turned. In true torticollis, the contracted muscle is on the opposite side. Hysterical cases are recognized by the sex and age of the patient and the ordinary concomitants of hysterical cases.

**Prognosis.**—In spasmodic torticollis this is very unfavorable. Temporary “cures” may be effected, but the trouble is liable to relapse.

**Treatment.**—Congenital torticollis finds a ready and efficient remedy in tenotomy of the affected muscle. This procedure is highly irrational in other varieties. Quite a variety of surgical measures have been suggested for spasmodic torticollis, and these have been followed with more or less success. Stretching the spinal accessory nerve is probably the one of first resort. In most cases, it at least produces a temporary stoppage of the spasm. Relapse is not at all unlikely. Excision of the nerve is followed by more permanent results, though of course the value of the operation is in a measure counterbalanced by the paralysis which necessarily ensues. As a rule the patient does not object to the inconvenience of this if he is thereby cured of his other infirmities. Physiological medication rarely if ever produces a permanent cure. Sedatives, as bromide of potassium, chloral, etc., exert no influence in genuine cases. Preparations of conium, taken in large and increasing doses, in many instances put a stop to the spasm temporarily. The movements return when the medicine is withdrawn.

Gowers recommends, but with considerable reserve, the use of morphia hypodermically, in doses gradually increased until from one-half to one grain is taken daily. Such treatment must be entered upon with considerable reluctance, because it is almost certain to set up the morphia habit.

Orthopædic apparatus designed to restrain the spasmodic movements is worse than useless. If the movement of one muscle is restrained, others take up the disordered action.

Electricity occasionally effects a cure. The faradic brush or static electricity is the best for hysterical cases. For others, applications of galvanism, the positive pole being applied to the motor points of the affected muscles, are indicated. The sittings must be frequently repeated, and continued over a long period of time. Erb also makes application of the positive electrode to the cerebral cortex of the opposite side. Sometimes he also passes the current transversely from the medulla to



the mastoid. Faradic applications to the affected muscles in the rheumatic variety of torticollis very often effect a rapid cure. Hot ironing of the muscles in these cases is a valuable adjuvant.

*Aconite*, *rhhus*, and *bryonia* are the remedies adapted to rheumatic cases. The former will often act curatively in recent cases of the spasmodic variety.

*Strychnia* is indicated in cases of tonic spasm, and will sometimes cure. I prefer the administration of grain doses of the second decimal trituration, every two to four hours. It and *nux vomica* are especially indicated in cases in which the association of spasms and causative indications suggest a spinal origin for the trouble.

*Gelsemium*, *cicuta*, *physostigma*, and *sulphur* are other remedies suggested. One must not build too high hopes on their action, however, in cases of the clonic spasmodic torticollis.

## AFFECTIONS OF THE HYPOGLOSSAL NERVE.

The hypoglossal nerve is purely motor, supplying the muscles of the tongue, and the depressors and some of the elevators of the hyoid bone. Lesions of it or its nuclei cause paralysis of these muscles, which, however, rarely exists unassociated with loss of power in other parts.

**Symptoms.**—In paralysis of one hypoglossal nerve, the tongue when at rest, presents a perfectly normal appearance aside from a slight elevation at its base on the paralyzed side. During muscular actions within the mouth, movement towards the diseased side is impaired. In protruding the tongue, the reverse condition obtains. When both hypoglossal nerves are completely paralyzed, the tongue lies immovable in the mouth. Articulation is affected only in bilateral paralysis, and then proportionate to the degree of loss of power. Paralysis of one half of the tongue, does not as a rule interfere with articulation. It does incommode the patient considerably during mastication, as the tongue is an important organ in enabling the patient to retain food between the teeth. The presence or absence of atrophy of the lingual muscles depends entirely upon the seat of the lesion. If between the nuclear cells and the cortex, no atrophy takes place. If the lesion is nuclear or infra-nuclear, atrophy is marked. The mucous membrane of the tongue is then thrown into folds and ridges.

**Diagnosis.**—The recognition of paralysis of the tongue is an easy matter. The only diagnostic difficulty, therefore, is found in the localization of the lesion. The first point of importance, the presence or absence of muscular atrophy, has already been mentioned. In addition, if the lesion is supra-nuclear, the motor tracts in the brain are involved, and there is hemiplegia on the same side as the glossoplegia. In nuclear paralysis, the lesion is nearly always bilateral; other nerves give evidence of loss of function; marked lingual atrophy is present. Nuclear lesions form a part of glosso-labial palsy and are occasionally observed during the course of locomotor ataxia and disseminated sclerosis. When the fibres of the nerve within the medulla are diseased, there is hemiplegia on the one side, and hemi-glossoplegia on the other. When the nerve is affected at its origin on the surface of the medulla, or in its course within the cranium, there is associated, paralysis of the corresponding halves of the palate and larynx.

**Prognosis.**—This depends entirely upon the nature of the lesion. As a rule, the prospects of cure, or even of improvement, are poor.

Inasmuch as paralysis of the tongue is but part of pathological

conditions described elsewhere, no special remarks concerning its treatment need be detailed, other than to specify the best method of electrical application. The negative electrode should consist of a small flat plate in the shape of a tongue depressor, with insulated handle. If galvanism is to be used, it must be covered with chamois skin or absorbent cotton. The positive pole should be applied to the nape of the neck.

**Spasm of the Tongue.**—Spasm of the tongue is of frequent occurrence as a part of general convulsions, as those of epilepsy and hysteria. It may exist as a purely hysterical manifestation, interfering with articulation. It is the cause of some varieties of stuttering. It sometimes arises from irritation in the area of distribution of the fifth pair of nerves. The spasm is generally paroxysmal, and may be either tonic or clonic.

**The Prognosis** is good.

The majority of cases will be found associated with general conditions, the treatment of which regardless of the existence of the spasm effects a cure.



# AFFECTIONS OF THE SPINAL NERVES.

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## NEUROMATA.

The term neuroma includes all varieties of tumor involving nerve trunks or their fibres. They may consist of an abnormal growth of nerve fibres, in which case they are called true neuromata, or of heterologous tissue, generally known as false neuromata. These latter consist of sarcoma, fibroma, syphiloma, and other varieties of tumor which may occur elsewhere.

The true neuromata consist of either medullated or non-medullated fibres, or even of ganglion cells. They may be single or multiple. Remarkable instances of the wide diffusion of numerous neuromata have been recorded.

The tumors are generally within the sheath of the nerve. Whether or not the nerve fibres are damaged depends upon their size and situation. Malignant growths, as sarcomata, are far more liable to give rise to suffering than are true neuromata.

**Symptoms.**—These may be entirely absent, the tumor itself being the only evidence of disease. In most cases, however, we find the usual symptoms attendant upon lesion of a peripheral nerve, pain and disturbance of sensation and motion. The pain may be of the mildest or most severe character; and the paralysis may range from slight weakness to complete motor loss. The tumor may be exceedingly sensitive or absolutely painless to rough manipulation. Sometimes they may be felt, but only when tolerably well-developed and in accessible situations. When numerous, they present bead-like swellings along the course of the affected nerve.

**Diagnosis.**—The symptoms presented are essentially those of neuritis, hence a diagnosis can only be certainly made when the tumor is felt. By exclusion of the special causes and more prominent conditions attending neuritis, one can make a tentative diagnosis of neuroma.

**Prognosis.**—This depends entirely upon the rapidity of the extension of the growth and the intensity of symptoms.

**Treatment.**—Medical treatment can do but little, excepting in the syphilitic varieties. Excision of the growth is only advisable when it produces symptoms, or is of a malignant character. Due consideration must be given the functions of the nerve in deciding as to the advisability of an operation.

## NEURITIS.

Neuritis is an inflammation of the structures of a peripheral nerve. It may affect the sheath of the nerve (*perineuritis*), the connective tissue binding together the separate fibres (*interstitial neuritis*), or the nerve elements proper (*parenchymatous neuritis*). One nerve only may be involved in the inflammation (*simple neuritis*), or the disease may be widespread, involving nearly every important nerve trunk in the body (*multiple neuritis*). According to the causes, neuritis has been divided into arsenical, alcoholic and plumbic neuritis, when arising from the ingestion of one of these poisons; post-febrile neuritis, occurring after certain fevers, such as typhoid fever, scarlatina, diphtheria, smallpox, etc.; malarial neuritis, when the result of malarial poisoning; syphilitic, leprous, or tuberculous neuritis, when a manifestation of one of these constitutional vices.

**History.**—Neuritis, as affecting single nerve trunks and occurring after injuries or exposure to cold, has long been an admitted fact. Multiple neuritis, caused by a profound poisoning of the system, is a disease concerning which completed knowledge has only been had within the past decade. Looking back over the literature of the past, we find reports of cases which in the light of present knowledge we now know to have been cases of multiple neuritis. In 1822, Dr. James Jackson, of Boston, described what is probably the first American case, under the title, "On a Peculiar Disease resulting from the Use of Ardent Spirits." Notwithstanding the antiquity of the report, one finds in it a graphic account of the clinical phenomena of multiple neuritis, not to be improved upon at the close of the century. Magnus, in 1852, in his great work on alcoholism, described the nervous symptoms of alcoholism under various headings, among which were included the paralytic and anæsthetic forms. This author made the mistake of assigning the lesions to the central nervous system. Lancereaux, writing in 1864, adopted the same conclusion. Duchenne, in his work, "Electrization Localisée," grouped these cases under the title, "Subacute Ascending Spinal Paralysis." In 1864 Dumenil made an autopsy which first established the actual existence of multiple neuritis. Two years later he reported another case with the post-mortem findings, whereupon he announced as his opinion that many cases hitherto obscure were due to neuritis of spontaneous origin. Eleven years elapsed before another observation was made, when Eichhorst reported a case, with not only the post-mortem findings, but the microscopical appearances of the diseased

nerves, still further establishing the true nature of the affection. In all of the instances thus far referred to, the interstitial variety of neuritis existed. In 1879 Joffroy reported a case occurring in a phthisical patient, the microscopical examination showing parenchymatous and not interstitial changes in the diseased nerves. Erb had, in the meanwhile, in Ziemssen's *Encyclopædia*, suggested the probability of many cases of neuritis being due to toxic influences. Since 1880 numerous cases of multiple neuritis have been described, until now the disease may be considered a well-established clinical entity. Our knowledge of it in America is largely due to Starr.

The different etiological factors at play in the production of simple and multiple neuritis make it convenient to describe these conditions separately.

### SIMPLE NEURITIS.

**Etiology.**—Prominent among the causes of neuritis of single nerve trunks is traumatism. The nerve may be divided by wounds, lacerated in fractures, compressed by dislocations, tumors, callus, or faulty postures. Very exceptionally, neuritis has followed compression from violent muscular action.

Isolated neuritis, like the multiple variety, may arise from constitutional causes, as syphilis, cancer, gout, lithæmia, leucocythæmia, and rheumatism.

Exposure to cold sometimes occasions a rheumatic neuritis. The best example of this we find in facial palsy following exposure of the face to a draught of air.

**Pathology and Morbid Anatomy.**—Microscopically, the first change observed in marked cases is swelling of the affected nerve trunk. Its sheath is injected and infiltrated. Exceptionally, the inflammation is of a suppurative character, it then arising by extension of similar inflammation from contiguous parts. Microscopically, the redness is discovered to be due to distension of the bloodvessels of the sheath, and these are found surrounded by leucocytes. Sometimes there may be minute hæmorrhagic extravasations. Secondary to these changes, which are found either in the nerve sheath or the interstitial connective tissue, the nerve elements proper suffer. The extent to which this takes place varies principally with the intensity of the inflammation, though the situation of the lesion likewise plays an important role. Thus if it be within a bony canal, where the nerve is surrounded by unyielding structures, the damage is far greater than elsewhere.

In parenchymatous neuritis the changes are essentially those observed in nerve degeneration. The white substance of Schwann breaks up into successively smaller and smaller segments, becoming cloudy and granular. At the same time the axis cylinder shows changes,



finally, in severe cases, disappearing. The nuclei of the nerve sheath increase in number. At last there remains scarcely more than the thickened nerve sheath. In very few cases, however, do these extreme changes occur.

In chronic interstitial neuritis the axis cylinders suffer but slightly. The nerve is diminished in size owing to changes in the white substance. New fibrous tissue results from the inflammation, thus causing cirrhosis of the nerve.

In syphilitic neuritis the changes are identical with those found in syphilitic inflammation of connective tissue elsewhere.

In cancerous neuritis, the inflammation involves the nerves by extension from adjacent malignant growths, or by reason of infiltration of the nerve tissues with cancerous cells.

**Symptoms.**—The onset of neuritis may be rapid or slow, according to the exciting cause and its intensity. Pain is generally the first symptom to attract attention. At first it is localized over the course of the affected nerve, and is greatly aggravated by pressure, and by influences which increase the tension on the affected nerve. Pressure on the nerve sometimes excites severe pain reaching to the points of distribution. Sensory disturbances are likewise prominent. These consist of numbness, tingling and anæsthesia in the regions supplied by the affected nerve. They may constitute the first symptoms, and in mild cases the only ones. They are generally experienced in the feet or hands. When the patient is thin, or the course of the nerve superficial, the inflamed and swollen structure may be felt. Constitutional symptoms are rarely prominent features in isolated neuritis. Only exceptionally do we observe a rise in temperature. Motor symptoms in the shape of paralysis are not likely to be obtrusive, unless the traumatism giving rise to the inflammation has been a severe one. In most cases, close examination reveals the existence of some loss of motor power.

The pain is not always strictly localized, since it may involve the entire limb. Muscles, skin and other structures become sensitive to touch.

Trophic symptoms of quite a variety appear. Local sweatings are observed. The skin becomes reddened and glossy. The muscles supplied by the inflamed nerve waste, and exhibit the reaction of degeneration. Fibrillary contractions are associated with the advancing muscular atrophy.

In the chronic form of neuritis the symptoms vary but little from those above enumerated. Pain is ever a prominent feature; indeed, the case may be regarded as one of neuralgia. The various trophic phenomena of neuritis are all present. In addition, the neighboring joints are apt to be affected, the articular inflammation showing a remarkable tendency to the production of adhesions.

**Diagnosis.**—The main reliance in the diagnosis of neuritis is to be placed on the localization of the symptoms, pain, weakness, paræsthesia, and anæsthesia, to an area tributary to certain nerve trunks. If these are kept in mind, there need be no danger of confounding neuritis with rheumatism, as is so often done.

The differentiation of *neuralgia* and neuritis is not so easy. The pain in the former is not so steady, and there is no tenderness along the course of the affected nerve, and, besides, the functional disturbances of the nerve are not prominent.

From *disease of the spinal cord*, neuritis is distinguished by the distribution of the symptoms.

**Prognosis.**—Traumatic cases present a favorable outlook, the great majority recovering with very nearly normal function. Suppurative neuritis rarely ends in recovery of function. In framing a prognosis one must be guided by the intensity of the symptoms and the extent of the degeneration as evidenced by the electrical reactions in the muscles tributary to the affected nerves. Even in favorable cases the course is a slow one. Advanced years lessen the chances of recovery.

### MULTIPLE NEURITIS.

**Synonym.**—Polyneuritis.

**Etiology and Pathology.**—Multiple neuritis affects principally persons in adult life, that is those between twenty and fifty years of age. Children are especially exempt. It not infrequently, however, attacks the aged. Cases arising from alcoholic excesses occur as a rule between the thirtieth and fortieth years. The predisposing influence of sex differs according to the cause of the trouble. The vast majority of cases of alcoholic neuritis occur in females; those arising from other agents, in males. Exposure to cold not infrequently excites multiple neuritis, but in such cases there must be a constitutional predisposition. In many, there must be a multiplicity of causes at work. Thus, in the case of a patient addicted to excessive indulgence in alcohol, exposure to cold and insufficient nourishment aid greatly in the production of the neuritis. The different causes impress their peculiar consequences upon the resulting attack.

A thorough examination of the clinical phenomena of multiple neuritis convinces one that a disease of this peculiar character can only arise from a widespread systemic poisoning. The lesions are remarkable for their symmetry, and their all but universal distribution. The more specific the character of the poison, the greater is this tendency to symmetry. Gowers lays down the maxim: Symmetrical multiplicity means a blood state acting alone; irregular multiplicity means a constitutional state combined with local determining causes. Some poisons possess the peculiar property of acting in a manifold manner in

the production of neuritis. Take, for example, alcohol. Its continued use may lead to gout, and this in its turn to an isolated gouty neuritis. The alcohol itself causes neuritis directly. Thus the alcoholic and gouty neuritis are combined in the same case.

Among the causes producing neuritis through the blood may be enumerated the following: Alcohol, arsenic, lead, bisulphide of carbon, rheumatism, gout, syphilis, tuberculosis; certain acute infectious diseases, notably diphtheria, variola, typhus and typhoid fevers, and septicæmia.

Experience at present at our disposal does not show that uncomplicated syphilitic neuritis is of frequent occurrence. It does occur quite frequently in conjunction with locomotor ataxia. Indeed, many undoubted cases of the latter affection reveal little, post-mortem, aside from a widespread neuritis. It has been claimed that certain anomalous symptoms of the disease, gastric crises, arthropathy, etc., are but the result of neuritis.

Tubercular neuritis occurs in the course of phthisis. It is believed to be the result of the specific action of the product of the bacillus of tuberculosis, for no bacilli have been found in the inflamed nerves. It is a common practice to administer large quantities of alcohol to phthisical patients, thus making it at times impossible to determine the part played by each factor in the production of the neuritis.

Cases arising from exposure to cold are classed as rheumatic. They generally present interstitial changes in the affected nerves.

Diabetes is quite a frequent cause of neuritis. Indeed it has been claimed that some degree of this complication is present in most cases. The quantity of sugar in the urine has no relation to the degree of neuritis. It is believed that the neuritis is the result of some toxic agent formed by the perverted chemical alterations going on within the body of the diabetic.

Atheromatous changes in the bloodvessels sometimes cause neuritis. The vascular supply of the nerve fibres is thus interfered with, and degeneration results.

Septicæmic neuritis has occurred secondarily to malignant endocarditis.

**Symptoms.**—In presenting in detail the symptomatology of multiple neuritis, it will probably conduce to clearness, if the cases resulting from alcoholism be taken as the type of all, after which the points on which neuritis arising from various causes differ, may be detailed. Alcoholic neuritis only sets in after long and excessive indulgence. Apparently, many cases set in rapidly, the patient stating that she was seized with paralysis in the midst of her duties. Careful examination, however, discloses that possibly for months before, she has complained of numbness, tingling, various paræsthesia, gastric symptoms, insomnia, tremor, peripheral pains, and increasing feebleness.



The paralysis begins, usually, in the lower extremities, often however affecting simultaneously both hands and feet. It spreads rapidly, but rarely extends to the trunk. The flexors of the foot and the extensors of the wrist suffer especially. Thus we find a very characteristic distribution of the paralysis which may be described as "dropped foot and dropped wrist." The affected muscles become flabby and undergo atrophy. They present the characteristic reaction of degeneration.

Tactile sensibility is abolished, as is the muscular sense in a great measure. This loss of sensation may be present only in irregularly limited areas; usually, however, it is complete over the entire distal part of the affected limbs.

Severe pains are often experienced. The affected parts are decidedly hyperæsthetic.

In the parts affected the deep reflexes are diminished or destroyed; the cutaneous reflexes are preserved.

Mental symptoms are prominent features of the alcoholic neuritis. These consist of delirium, illusions, hallucinations, insomnia, loss of memory, and defective power of concentration. Strümpell has called attention to the disposition on the part of some patients to relate as actual facts stories purely the product of the imagination.

Alcoholic neuritis reaches its acme shortly after its onset, after which it remains stationary for a while. Then the symptoms recede. The mental symptoms above enumerated afford valuable aid in recognizing the cause of the trouble. Unfortunately, their value is lessened by the fact that they do not appear until the neuritis has far advanced. Buzzard calls attention to a symptom not mentioned by any other authority, a symptom that is of differential diagnostic value if future experience shall show him to be right. In women suffering from alcoholic neuritis, there is an associated amenorrhœa, and as the neuritis improves, the menstrual flow reappears. Another condition observed by the same authority is an urticaria with purpura-like hæmorrhages, affecting especially the lower extremities. As bearing on the recognition of alcoholic neuritis may be borne in mind certain pathological phases of the disorder; since almost invariably one in bad cases finds evidences of serious degeneration throughout the central nervous system, brain and spinal cord being alike affected. These changes are not limited to certain structures, but are widely disseminated.

*Arsenical neuritis* is characterized especially by the prominence of the motor symptoms.

*Neuritis from lead* affects principally the musculo-spiral nerves.

Reference to the neuritis arising in the course of the various infectious diseases will be found in the sections devoted to the consideration of these affections.

Endemic neuritis and beri-beri will receive separate consideration later.

**Diagnosis.**—There can be no doubt, if we are to decide by clinical data, that many obscure minor affections hitherto classed under the names numb fingers, paræsthesia neurosis, etc., are really instances of mild neuritis. Many others are examples of vaso-motor neuroses of which Raynaud's disease may be taken as the type.

Severe cases of neuritis very closely simulate certain spinal affections. From *poliomyelitis anterior* it is differentiated by the presence of pains and sensory disturbances, the mode of onset, the age of the patient, the strictly symmetrical distribution of the paralysis and the relation of the muscles paralyzed to definite nerve trunks. In poliomyelitis the paralysis reaches its height at once; in neuritis it increases rapidly for several days.

The resemblance of neuritis to *ataxia* is often quite puzzling. The history of the case is usually sufficient to decide. Should this not be so however, one can on close examination detect certain characteristic differences in the gait. In both affections the patient lifts his feet high from the ground when stepping. In *ataxia*, the toes are elevated, and when the feet are brought to the ground, the heel comes down first, the toes following. In neuritis, there is dropped foot, and the toes reach the ground first, the heel following. Neuritis may be still further distinguished by the absence of lightning pains and bladder symptoms, and the presence of the reaction of degeneration. In *ataxia* the disordered gait is of relatively slow onset.

*Subacute and chronic spinal paralyses* are manifested by the symptoms of poliomyelitis pursuing a slow course. Hence these are readily recognized by the absence of pain and the distribution of the palsy. *Diffuse myelitis* is distinguished by its involvement of the sphincters, the presence of the girdle sensation, the extension of the paralysis to the trunk, the bedsores and cystitis.

**Prognosis.**—As a rule, if proper treatment is instituted, cases of multiple neuritis end in recovery. The course of the disease is a slow one, ranging from two to fifteen months. Sometimes permanent contractures and deformities occur. Some cases end fatally.

## BERI-BERI.

**Synonyms.**—Kakke; endemic neuritis.

**History.**—Beri-beri has existed in certain countries from most remote ages. Definite records of its prevalence in China two hundred years before the Christian era are to be had from the ancient literature of that country. About two hundred years ago it disappeared from China for reasons not understood. Its ravages are now largely confined to Japan, though it appears not infrequently in the islands of the Pacific Ocean, India, Ceylon, the west coast of the Red Sea, New Guinea, Brazil and Cuba. The attention of Europeans was first directed to the disease

by Prof. Scheube, of Tokio, Japan, in 1882. Its prevalence in Japan can be well appreciated when it is stated that in 1877, 14 per cent., and in 1878, 38 per cent. of the men in the army service contracted the disease. Beri-beri seems to be an infectious miasmatic disease. It bears, apparently, some relation to rice-eating, for since wheat was substituted for rice in the army dietary list, the disease has greatly declined in frequency. The great epidemic of 1882-3 in Manilla followed a period of six months in which the populace subsisted almost exclusively on rice, a diet adopted by them in consequence of the prevalence of cholera. In 1881, and again in 1889, there were epidemics of a disease closely simulating beri-beri among the crews of fishing vessels along the Newfoundland coast.

Beri-beri attacks almost exclusively the young and strong, those, by preference, between the ages of sixteen and twenty-five. Females are almost entirely exempt from it. Overcrowding, as in barracks and camp, exposure to dampness and cold, strongly predispose to it. The disease prevails most extensively in hot weather. Europeans are almost entirely immune.

The disease has been proven to be due to a special bacillus, though the methods by which it is propagated are as yet unknown.

**Symptoms.**—The symptoms of beri-beri vary greatly, according to the severity of the case. For descriptive purposes we may divide cases into mild and severe. The mild are characterized by mild fever and catarrhal symptoms, succeeded by slight motor and sensory disturbances. The patient notices himself to be weaker than usual, and complains of peripheral pains and numbness, especially in the legs. With these symptoms there may be slight œdema, cardiac oppression, loss of appetite and other indefinite symptoms. Physical examination of these cases shows a slight degree of the reaction of degeneration, some loss of muscular power and anæsthesia.

The severe cases may be described as being of three types: (1) The atrophic or dry type; (2) the hydropic or wet type, better called the œdematous; (3) the acute pernicious type.

(1) **THE ATROPHIC OR DRY TYPE.** The symptoms are the same in general character as those detailed in the mild cases, but are far more severe. The pains are atrocious; the muscular weakness proceeds to complete paralysis; muscular atrophy becomes extreme until finally the patient is but little more than a living skeleton; the reaction of degeneration is typical.

(2) **THE HYDROPIK OR WET TYPE.** The symptoms of this are the same as those of the atrophic type, with the addition of œdema and circulatory disturbances. The cardiac weakness is probably the cause of the dropsy.

(3) **THE ACUTE PERNICIOUS TYPE.** This combines the symptoms of



the preceding types, which pursue their fatal course with a peculiar malignancy.

**Prognosis.**—This varies with the epidemic and the character of the attack. Mild cases recover. Those of the atrophic type generally make complete recoveries, though only after months of disability. The majority of the acute pernicious type die of heart-failure within two weeks from the onset of the disease.

**Treatment of the Various Forms of Neuritis.**—The first essential is absolute rest in bed, if possible; if not, then as nearly entire rest of the part in which the nerves are inflamed as can be had. It is the duty of the physician to give the patient to distinctly understand that any exercise, even the slightest, in any case of neuritis, isolated or multiple, is injurious to his welfare; to comfort, when isolated nerves are inflamed; to comfort and life, when the disease is multiple. When a nerve of the arm is affected, that limb should be carried in a sling. The patient should not think of performing with the afflicted member even such simple acts as dressing or feeding himself.

The diet must be plain and unstimulating.

Great care should be exercised in learning the cause of the trouble, for if this is allowed to remain active, recovery need not be expected.

In isolated neuritis, causes acting locally must be sought for.

For the relief of pain, hot or cold applications, according to the peculiarities of the case, should be employed in preference to analgesics. As a rule, heat is the most acceptable to the patient, and may be applied by hot cloths, hot-water bags, or the Japanese hot-box. I most certainly prefer the latter, when the heat is to be localized upon a small area, and the weight of the hot-water bag is objectionable. In the use of hot applications one must bear in mind the anæsthesia and the danger resulting from making them of too high a temperature.

Some cases reject heat as unbearable or as apparently injurious, and obtain benefit from ice-cold applications. Probably the best means of applying cold will be found in the use of a tin box, one side of which is so formed as to take the shape of the affected limb. When these measures and remedies fail to give the desired relief, then and then only should palliative medication be sought. Morphia is probably the most reliable, especially when given hypodermically. The duration of the illness is likely to be such, however, as to make the possibility of producing the morphia habit a matter for serious consideration. I myself much prefer acetanilid in from five to ten grain doses repeated with due caution, and only when absolutely necessary. Under no circumstances would I advise its use without most careful attention to its effects on the heart. Nor would I ever resort to maximum dosage without first determining the patient's susceptibilities to smaller ones.

Alcoholic cases require absolute removal of all alcoholic beverages,

excepting when the patient is so debilitated as to make the sudden withdrawal of the accustomed stimulus unsafe. These patients require especial attention as regards nutrition. Koumiss, peptonized milk, beef tea, and other easily digested and nourishing articles should be given in small quantities and at short intervals. If the stomach rejects food, it must then be administered per rectum. Sometimes the use of red pepper in the beef tea, as in other alcoholic diseases, is beneficial.

Electricity is a most valuable adjuvant. The faradic current is valueless, if not harmful, as it interferes with rest of the parts. The proper method consists in the use of galvanism, a mild current without interruptions, for about five or ten minutes daily. The positive electrode should be applied over the seat of greatest pain, and the negative over the spinal origin of the nerve affected, or to the periphery of the extremity, hand or foot as the case may be. The current should be gradually increased and gradually diminished, carefully avoiding any shocks or interruptions. It is unwise to resort to electricity early in the course of multiple neuritis. Withhold the treatment until active symptoms have subsided. *For the restoration of the atrophied muscles* galvanism and massage are the main remedies. The current for this purpose should be an interrupted one, and of just sufficient strength to excite muscular contraction. The massage should be administered by a professional masseur. To rely upon the well-meant though misdirected efforts of the relatives or friends of the patient is the means of losing much valuable time.

Beri-beri requires in some instances a change in the diet, and always when possible the removal of the patient to a non-infected district. This in many cases is followed by rapid improvement in the symptoms.

For traumatic cases *arnica* and *hypericum* are the leading remedies. The former is the better in cases in which the lesion is the nature of a bruise or compression; the latter when from a laceration or wound.

Rheumatic neuritis, indeed most cases of perineuritis and adventitious neuritis, find their most frequently indicated remedy in *rhua*. This remedy is especially useful in the subacute and chronic cases. I prefer the administration of the first decimal dilution, one drop every one to three hours. I do not think that I have obtained very good results from the dry preparations of this remedy.

Acute cases require *aconite* or *ferrum phos.*, according to the character of the inflammation. Aconite is preferable when the numbness is a prominent feature, and when the trouble has arisen from exposure to cold.

For the severe cases in which pains are atrocious, no remedy is better indicated than *belladonna*, although when these are of a severe burning character, and motor paralysis is pronounced, *arsenicum* is also invaluable.

In multiple neuritis following acute infectious diseases, one can rely mainly on *gelsemium*, *argentum nitricum*, *causticum*, and *rhus*. I prefer the former two in the majority of cases; the *gelsemium* in the paralytic, and the *argentum nitricum* in those of ataxic type. *Bisulphide of carbon* may be suggested as of possible use in the latter cases.

In the alcoholic cases, I prefer *nux vomica* 1x. If the active symptoms have subsided, and prostration is profound, *strychnia* 2x. O'Connor relies mainly upon *cimicifuga* in these cases. His indications for the remedy are: Aching pains in the limbs, which the patient likens to a toothache, and the use of alcohol in any form as a causal factor. He gives one drop of the tincture to four ounces of water, in teaspoonful doses, repeated every few hours.

*Arsenicum* is probably the remedy best adapted to the treatment of severe forms of multiple neuritis.

In the atrophic stage of all forms of neuritis, *plumbum* is the best remedy.

*Rhus* and *causticum* are suggested in paralytic cases with the subsidence of all inflammatory symptoms.

*Phosphorus* is well adapted to cases of degenerative neuritis. It may be given either in dilutions of the pure substance or in triturations of phosphide of zinc.

Kenneth McLeod has proposed and put in practice in eight cases, the longitudinal section of nerves affected with interstitial inflammation, leprous and otherwise. Two of these cases were promptly cured; four were greatly benefited, and in two the treatment was a failure. These results are very good when the obstinate nature of the malady is considered.



## DISEASES OF THE CERVICAL PLEXUS.

**Cervico-occipital Neuralgia.**—This is a rather common form of neuralgia. It is frequently met with in women, with whom it is often symptomatic of hysteria or pelvic trouble. It may arise from cold, as exposure of the back of the neck to a draught. It is at times a symptom of vertebral caries. Cervico-occipital pain has been met with as a result of refractive errors, and insufficiency of the ocular muscles.

The pain involves part of or the entire territory supplied by the first four cervical nerves. Usually, the great occipital nerve only is affected. In severe cases the pain may extend so far forward as to give the impression of an involvement of the trigeminus; and at other times it reaches to the shoulders and even into the arms. It is sometimes associated with torticollis. The discovery of the cause of the neuralgia can only be made through a study of the associated symptoms.

The PROGNOSIS is essentially that of the primary cause. Usually these cases are readily curable. Those depending upon vertebral caries offer an unfavorable prognosis.

The TREATMENT must be symptomatic and adapted to the causative conditions of the neuralgia.

**Paralysis of the Branches of the Cervical Plexus** is sometimes observed in the course of progressive muscular atrophy, cervical pachymeningitis, vertebral caries and injuries. Paralysis of the neck muscles results.

**Paralysis of the Phrenic Nerve.**—The phrenic nerve is generally regarded as a motor nerve, though some think otherwise. Peter, for example, has described what he has termed *phrenic neuralgia*, a condition characterized by severe pain along the lower and anterior part of the thorax over the line of attachment of the diaphragm, and along the course of the phrenic nerve.

The motor disorders of the phrenic nerve are paralysis and spasm. The former may be due to disease of the cervical portion of the cord, or to disease or injury of the phrenic nerve in its long course. The result of either of the above-named lesions is paralysis of the diaphragm, alone if the phrenic nerve is injured or diseased; in conjunction with paralysis of other muscles if the disease be spinal. The phrenic nerve may be injured by wounds of the neck, or it may be compressed by intra-thoracic tumors. The nerve may be inflamed by cold, or as a part of an attack of multiple neuritis, as after diphtheria or other infectious diseases, or during the course of lead poisoning. Diaphragmatic paralysis may be

either uni- or bi-lateral. It is recognized by the absence of movement of the abdominal muscles during respiration, and retraction of the epigastrium and the hypochondria during inspiration. Respiration is carried on by the intercostal and accessory respiratory muscles. Respiratory defect is not apparent while the patient is at rest. Very slight exertion, however, serves to produce rapid respiration, and even dyspnœa. Cough is rendered difficult. Inflammatory affections of the respiratory apparatus are thus rendered peculiarly dangerous. Unilateral diaphragmatic paralysis is shown by the presence of the above signs on one side only.

**DIAGNOSIS.** When paralysis of the diaphragm is associated with paralysis of other muscles, showing disease in the cervical portion of the spinal cord, the diagnosis is an easy matter. Some difficulty is occasionally encountered in distinguishing diaphragmatic palsy from the nervous breathing of hysterical females. Careful prolonged observation of the respiratory movements, while distracting the patient's attention, usually solves the problem. Movements of the diaphragm finally take place and the diagnosis is settled. In inflammation of the diaphragm movements of that muscle are restrained. The diagnosis is made by the presence of inflammatory symptoms, or the history of pleurisy or peritonitis.

**PROGNOSIS.** Paralysis of the phrenic nerve is always a serious matter. It is often fatal.

**TREATMENT.** This, in general, is the same as already outlined for neuritis. Galvanism of the phrenic nerve is of service. One electrode should be placed directly outside the lower portion of the clavicular portion of the sterno-mastoid, and the other upon the epigastrium.

**Spasm of the Phrenic Nerve.**—Spasm of the diaphragm may be either tonic or clonic. Tonic spasm occurs almost exclusively in conjunction with tetanus, and is the cause of death in that disease. It is sometimes observed in tetany. It very rarely, indeed, occurs as an idiopathic condition, and is then believed to be a rheumatic affection of the diaphragm. Treatment to be efficient must be prompt, as this condition occasions death in a very few minutes. The best measures are hot fomentations, the faradic brush to the region of the diaphragm, and inhalations of chloroform.

**Clonic Spasm of the Diaphragm** is generally known as *hiccough* or *singultus*. This is a very common trouble, and usually a trivial one. It sometimes proves very obstinate, and then becomes distressing if not actually serious. It has been observed in the course of organic diseases of the central nervous system, and is not infrequent as an hysterical phenomenon. Its most frequent cause, however, is reflex irritation from the abdominal viscera. Thus it occurs from overloading or disorder of the stomach, and from gastric, hepatic, intestinal and uterine diseases.

It is a serious symptom in the course of peritonitis. It is a terminal symptom of many incurable conditions, as uræmia.

Mild cases can be promptly cured by drinking water, strong emotional excitement, and numerous other contrivances well known to domestic medicine. Serious cases often require energetic treatment. In some, washing out of the stomach is followed by a prompt cure. Cruveilhier suggested the pouring of water into the mouth until the patient fears that he is about to be suffocated. The faradic brush over the diaphragm or hypochondria is often very efficient. In hysterical cases, hypodermic injection of one-twentieth of a grain of apomorphia by producing vomiting generally brings the paroxysm to an end. In obstinate hysterical cases, isolation from home and friends is essential to recovery.

As to remedies, *ignatia*, *nux vomica*, *cicuta*, *stramonium*, *arsenic*, *pulsatilla*, *hyoscyamus*, *veratrum album*, and *creasote* may be studied.



## DISEASES OF THE BRACHIAL PLEXUS.

The brachial plexus is derived from the last four cervical and the first dorsal nerves. Its ultimate branches are distributed to the muscles and integument of the upper extremity. Paralysis in this territory, as a result of nerve lesion, may be limited to the muscles supplied by a single nerve, or distributed over the course of several nerves. These paralyzes present all grades of severity, ranging from a slight numbness and tingling associated with a mere weakness which lasts for but an hour or two, to a complete destruction of motor and sensory functions.

**Combined Paralysis of the Nerves of the Arm** is the expression used to designate a condition in which more than one of the nerves of this member are affected by injury or disease. It may arise from disease or injury in the brachial plexus itself, pressure of morbid processes in the neck on the nerve trunks, to nerve injury, or to neuritis. Among injuries, those arising from shoulder dislocations, especially of the sub-coracoid variety, are the most common. Paralysis from this cause may involve all the muscles of the arm. These cases must always be looked upon as serious ones, for the nerves are subjected to considerable compression. Atrophy and paralysis are often extreme.

**Erb's Paralysis** is a combined palsy, in which the deltoid, biceps supra-spinatus, infra-spinatus, brachialis anticus and supinator longus are paralyzed. The nerves involved in these cases are the fifth and sixth cervical. The patient cannot raise the arm at the shoulder, nor can he flex the forearm on the elbow. The movements at the wrist and fingers are unimpaired; and the arm flexed at the elbow can be extended through the agency of the unaffected triceps. This form of paralysis may arise from injuries, neuritis, or the pressure of morbid growths. It is not infrequently observed as the result of injury during birth.

A lower arm type of palsy has been described. It is characterized by paralysis of the triceps, the flexors of the wrist, the pronators, the flexors and extensors of the fingers, and the hand muscles. It is due to lesions of the seventh and eighth cervical and first dorsal roots.

*The musculo-spiral and ulnar nerves* are sometimes injured simultaneously in fracture of the humerus.

In fracture of the forearm *the median and ulnar nerves* are the ones likely to suffer together.

**The Prognosis** in combined palsies varies according to the cause of the trouble, the severity of the lesion and the promptness with which treatment is instituted. Paralysis from dislocation of the shoulder gen-

erally makes a good recovery, if reduction is made promptly and properly. Cases of Erb's paralysis resulting from injury during birth offer a favorable prognosis.

Recovery may be tedious, and not complete for a year. Cases of paralysis due to laceration of the nerves and separation of the divided ends offer an unfavorable prognosis without operation.

**Paralysis of the Long Thoracic Nerve.**—This nerve supplies the serratus magnus. It is subject to quite a variety of traumatisms in its course in the neck. It is most frequently injured, however, by compression, as in carrying heavy weights on the shoulder, or by muscular action with the arm elevated, as in whitewashing a ceiling. It can occur as a result of cold. Paralysis of the serratus is observed also as a part of general palsies, as progressive muscular atrophy and poliomyelitis anterior. The recognition of paralysis of the serratus magnus is made through the position of the scapula, the inferior angle of which is nearer to the vertebral column than normal; at the same time the posterior border of the bone projects, giving it an appearance that has been described as "winged."

**Paralysis of the Circumflex Nerve.**—This nerve supplies the deltoid and teres minor muscles, and the skin over the deltoid. It is very liable to injury from blows and falls, and to compression from dislocations and crutch pressure. Its principal symptom is inability to raise the arm. Some slight motion of the deltoid is preserved in its anterior portion, which derives its nerve supply from the anterior thoracic nerve. The atrophy of the deltoid causes important changes in the shape of the shoulder, which becomes flattened. The ligaments of the shoulder joint become relaxed, and a concavity forms beneath the acromion process.

**Paralysis of the Musculo-cutaneous Nerve.**—This nerve supplies the biceps and the brachialis anticus muscles and the skin over the radial side of the forearm. The condition exhibiting its loss of function can therefore be surmised. It is rarely paralyzed alone.

**Paralysis of the Musculo-spiral Nerve.**—Owing to its position as it winds around the humerus, this nerve is very liable to suffer from injuries, especially from compression. More frequently than from any other cause, it is injured by sleeping with the head resting upon the arm. Unless the sleep be part of a debauch, and consequently a very deep one, the resulting paralysis is of very short duration. Most cases coming to the physician's attention, however, occur during a drunken stupor, and are apt to last for several weeks. The paralysis affects the extensors of the wrist and fingers and the supinator longus. If the nerve is injured high up in the arm, then the triceps is also paralyzed.

Paralysis of the extensors of the wrist is a very common symptom of neuritis from lead poisoning. In such cases, the trouble is almost

always bilateral, and there is a history of other manifestations of lead poisoning.

**Paralysis of the Ulnar Nerve.**—This nerve supplies the flexor carpi ulnaris, the inner part of the flexor profundus digitorum, the muscles of the little finger, the interossei, the third and fourth lumbricales, and the adductor and inner head of the short flexor of the thumb. It supplies sensation to the ulnar side of the hand, generally more extensively on the back than on the front. It may suffer from neuritis or injury. Paralysis in its distribution may result from prolonged flexion of the elbow. The symptoms are as follows: "The hand deviates to the radial side, owing to paralysis of the ulnar flexor of the wrist. Flexion of the first phalanges is impossible, and also adduction of the thumb." In old cases the first phalanges become hyper-extended, while the others assume a position of strong flexion. The hand becomes claw-shaped, the so-called *main en griffe*.

Dana describes what he calls "A SYMMETRICAL SPONTANEOUS ULNAR NEURITIS," a condition which develops slowly in neurotic subjects without known cause. It is characterized by pain and paræsthesia in the parts supplied by one ulnar nerve, with subsequent deformity and extension to the other side. He assigns degenerative neuritis as the pathological cause. Mild cases of this trouble are doubtless very common.

**Paralysis of the Median Nerve.**—This nerve supplies the pronators, the flexor carpi radialis, the flexors of the fingers with the exception of the ulnar half of the deep flexor, the flexors and abductors of the thumb, and the first and second lumbricales. When the median nerve is paralyzed, pronation beyond the mid-position is impossible; flexion at the wrist is still possible, but only with strong deflection of the hand towards the ulnar border of the forearm. The thumb rests extended and adducted. The second phalanges cannot be flexed on the first; and the distal phalanges of the first and second fingers on the middle phalanges. Flexion of the distal phalanges of the third and fourth fingers is still possible by means of the ulnar half of the flexor profundus digitorum.

**Brachial Neuritis.**—This is a not uncommon affection, in which the sheaths of the cords of the brachial plexus, or of the roots of the spinal nerves feeding the same, are inflamed. It may be either primary or secondary. In the latter case, it usually originates in an ascending neuritis starting in one of the nerves of the arm. Primary brachial neuritis occurs as a manifestation of the gouty diathesis, especially in females who have passed the age of fifty. It can occur from malarial poisoning.

**SYMPTOMS.** The prominent symptom of brachial neuritis is pain in the arm. The motor functions of the limb, *per se*, seem to be but little impaired. Whatever muscular weakness exists appears to be due rather



to the exquisite sensitiveness of the parts than to any paralysis. The muscles are usually soft and flabby, and but rarely atrophied. The seat of pain varies. In some cases it is quite peripheral, that is in the forearm and hand, while in others, it is attributed to the brachial plexus itself. When the spinal roots themselves are affected (radicular neuritis), there are severe pains about the spine. The pains are intense in character, almost continuous, and aggravated by the slightest touch. They usually follow the course of the nerves of the arm, but may be quite diffused. In mild cases, the pain occurs in paroxysms only. Anæsthesia of the skin sometimes occurs, especially in radicular neuritis.

**DIAGNOSIS.** Differentiation from neuralgia and angina pectoris is difficult, indeed often impossible. From neuralgia, brachial neuritis is to be distinguished by the persistent tenderness of nerve trunks, the influence of movement on the pain, and the history of the case. Severe brachial pains on the left side are apt to have the same distribution as the pains of angina pectoris, and to be associated with cardiac distress. The arm pains are primary, the heart symptoms reflex or secondary. Many obstinate arm pains are attributable by practitioners to compression from tumors or extension of organic disease within the chest. Such errors may be guarded against by careful physical exploration, and the knowledge of the existence of such an affection as brachial neuritis. By the very careless, this complaint is usually diagnosed "muscular rheumatism."

**PROGNOSIS.** Brachial neuritis is apt to be a tedious malady. It often runs a course of months. Relapses and recurrences are common. Advanced age makes the prospects of a good recovery smaller. A serious sequel of brachial neuritis is alterations in the joints of the arm. These undergo an adhesive inflammation with succeeding deformity.

The **TREATMENT** is the same as that for neuritis generally.

**Digital Neuralgia** is sometimes observed as an apparently idiopathic condition. It is usually traumatic in origin. Sometimes it occurs as a reflex symptom.

# DISEASES OF THE LUMBAR AND SACRAL PLEXUSES.

## THE LUMBAR PLEXUS.

The lumbar plexus is derived from the first three and part of the fourth lumbar nerves. Its branches are the ilio-hypogastric, ilio-inguinal, genito-crural, external cutaneous, anterior crural, and obturator nerves. These are subject to the usual mishaps which may befall peripheral nerves. The plexus itself may be damaged by intra-abdominal growths, especially those originating in or about the lumbar glands, and suppurative inflammations in this locality.

**The Anterior Crural Nerve** is the most important branch of the lumbar plexus. It supplies the psoas, iliacus internus, quadriceps extensor, sartorius, and pectineus muscles. All of these are paralyzed only when the nerve at its origin from the plexus is injured. Then flexion of the thigh on the abdomen is impossible. Disease within the abdomen involving the nerve trunk itself impairs flexion at the thigh, because of the paralysis of the iliacus. Lesion of the nerve after the giving off of the branch to the last named muscle, causes paralysis of the quadriceps extensor, the pectineus and the sartorius. The involvement of the latter two occasions no symptoms. The paralysis of the quadriceps makes extension at the knee impossible. Walking is thus seriously interfered with. The knee-jerk is destroyed. The accompanying anæsthesia covers the thigh with the exception of a narrow band posteriorly, and the inner side of the leg and foot.

NEURALGIA OF THE ANTERIOR CRURAL NERVE is rare as an idiopathic affection. It is usually symptomatic of morbid growths about the spine, or within the abdomen. It is sometimes due to neuritis starting in the sciatic nerve and extending to the lumbar plexus through the lumbo-sacral cord. The pain occupies the area above-mentioned as being anæsthetic, but it is especially severe along the course of the nerve.

The anterior crural nerve may be involved by injuries, psoas abscess, hip-joint dislocation, and disease of the vertebra.

**The Obturator Nerve** is especially liable to paralysis from injury during parturition. There follows a loss of power of adduction of the thigh, and the patient is unable to throw one leg over the other. Outward rotation of the thigh is impaired.

The remaining branches of the lumbar plexus, the *ilio-hypogastric*, *ilio-inguinal*, *genito-crural* and *external cutaneous*, may be the seat of very obstinate primary or secondary neuralgiæ. According to the nerve or nerves affected, the pain is distributed to the gluteal, hypogastric, or

genito-crural region or to the outer side of the thigh. Irritation of the ilio-inguinal nerve is believed to be the cause of the so-called irritable testis, a painful affection described by Cooper. It is very apt to be accompanied by a faint sickening feeling similar to that arising when the testicles are compressed.

### THE SACRAL PLEXUS

The Sacral Plexus is derived from part of the fourth and all of the fifth lumbar, and from the first to the fourth sacral nerves inclusive. Its branches are the great and small sciatic nerves and the pudic. The sacral plexus is very liable to involvement in intra-pelvic growths, inflammations, and suppurations. Indeed, every obstinate case of pain or paralysis in the territory supplied by its branches calls for thorough and painstaking physical exploration of the pelvic contents, under ether if necessary.

**The Small Sciatic Nerve** is distributed to the gluteus maximus and the skin of the back part of the thigh and leg. It is rarely involved apart from other nerves. The paralysis of the gluteus maximus interferes mainly with rising from a sitting posture, rather than with walking. The anæsthesia is in the area already indicated.

**The Great Sciatic Nerve** supplies the flexors of the leg on the thigh and all the muscles below the knee. When the lesion of the nerve is situated below the middle third of the thigh, the flexor muscles escape, and the paralysis is confined to the leg muscles entirely. Walking is not as much interfered with in this form of paralysis as one might imagine. The limb is held rigid at the knee by the quadriceps extensor. In walking, the limb is lifted by what may be called hyper-flexion at the hip. The anæsthesia involves the outer half of the leg, and the greater part of the dorsum and all of the sole of the foot. Neuralgia of this nerve is called sciatica, and is of sufficient importance to require a separate section of this work for its presentation.

The branches of the great sciatic nerve are the external popliteal or peroneal, and the internal popliteal.

**The External Popliteal or Peroneal Nerve** is the motor nerve of the tibialis anticus, the long extensor of the toes, the peronei, and the extensor of the little toe. Its paralysis gives the phenomenon so graphically described as "dropped-foot." As the ankle cannot be flexed, the whole leg is lifted higher than normal in walking, the toes at the same time drooping. This is called the "steppage gait." The anæsthesia covers the outer half of the front of the leg and the back of the foot.

**Paralysis of the Internal Popliteal or Posterior Tibial Nerve** destroys the function of the popliteus, the calf muscles, the tibialis posterior, the long flexors of the toes, and all the muscles of the sole of the foot. Sitting with the leg flexed at the knee, the patient is unable to



rotate the foot inward. Extension of the foot at the ankle joint is impossible. The position is that of talipes calcaneus. In disease of the *external plantar nerve* the toes become flexed at the last two joints and extended at the others. This position interferes very materially with walking. The accompanying anæsthesia is on the outer side of the sole of the foot.

**Erythromelalgia.**—This is an affection the character of which is not as yet understood, and which was first described by Mitchell in 1872. It consists of severe pains in the extremities, accompanied by congestion of the affected part. It has been variously attributed to spinal, vaso-motor and neuritic changes, the probabilities in our present light being in favor of its peripheral origin. The majority of cases occur in men. The congestion of the painful part does not appear until it is made to hang down, when it at once assumes a rose-red color. The arteries of the part throb violently, and then the color becomes darker and darker until it is finally of a purple tint. The pain is always aggravated at such times. In bad cases it may be constant; in mild ones it is only present when the part is dependent. It is worse in summer and better in winter. In some cases it prevents walking entirely. There is no diminution of sensation. The prognosis of the disease is unfavorable. According to Mitchell, bad cases do not recover. The only treatment that seems to be efficient is exsection of the nerves going to the foot. This can be done without much detriment to the patient, for they subserve unimportant motor functions.

**Morton's Neuralgia; Metatarsal Neuralgia.**—This is a form of neuralgia the pain of which starts at the base of the fourth toe. If at all severe, it radiates into the leg. It is aggravated by pressure over the seat of pain, or by squeezing the foot. It is believed to be caused by a neuritis, the result of bruising of the nerve by the fifth bone. Some claim that it is due to a slight luxation, and still others, to incipient flat foot. It is more common in women than in men. Its diagnostic symptom is the aggravation of pain in the locality above named by lateral pressure on the foot. Mild cases yield to the adoption of a proper shoe, *i.e.*, one which permits of no lateral pressure on the foot. Severe cases sometimes require excision of the head of the fourth metatarsal bone, and even amputation of the toe.

**Tarsalgia; Policemen's Disease.**—This is a form of neuralgia of the foot variously attributed to flat foot, stretching of the plantar ligaments, or contusion of the soft parts over the heel, brought on by excessive walking or standing. In the presence of a continuance of the exciting cause, treatment avails but little. Broad shoes with india-rubber heels have been recommended.

**Reflex pains** in the feet sometimes occur. They originate more frequently than from other causes, in disease in the course of the genito-urinary tract.

**Neurasthenic pains** are often known to affect the feet.

## SCIATICA.

Originally the term sciatica was intended to convey the idea of a neuralgia affecting the sciatic nerve. Extended experience in practice and pathological study have shown that nearly all these cases of so-called neuralgia are in reality instances of inflammation or other morbid changes affecting the sciatic nerve or its neighboring tissues. At present, therefore, sciatica includes all cases of pain referred to the sciatic nerve.

**Etiology.**—Males are more frequently the subjects of sciatica than are females, in the proportion of four to one. This is shown by the statistics of Gowers, Dana and others. It is without doubt due to the greater exposure of males to the exciting causes of sciatic neuritis. Age is a powerful predisposing cause of sciatica. Children are never affected, and adolescents only exceptionally. It is most frequently observed in persons between the ages of forty and fifty years. Among constitutional predisposing causes, rheumatism, gout and syphilis are prominent. Most authorities give syphilis a minor position in the causation of sciatica. Proper clinical investigation, however, will show many cases to thus arise.

The prominent exciting causes are exposure to cold, mechanical injuries, and muscular exertion. The exposure to cold is often local. Draughty water-closets have been credited with thus producing the trouble. This affords us an important suggestion respecting the management of sciatica. Cases arise from mechanical causes, as the result of compression from habitual faulty positions while working, and at other times.

Many cases of what appear in the beginning to be simple sciatica, are later found to be dependent upon morbid growths within the pelvis, disease of the pelvic bones or hip-joint, vertebral caries or cancer, spinal cord disease, and organic disease in the tissues surrounding the sciatic nerve in any part of its course. Toxæmic conditions which so frequently produce multiple neuritis, may be causative of sciatica. A notable example of this is found in diabetes. The sciatica in such cases is usually bilateral, and careful examination finds that other nerves than the sciatic are also implicated.

**Pathology.**—As already outlined in the remarks on etiology, the principal pathological change in sciatica is found in an inflammation of the nerve sheath. Such cases are for convenience called primary sciatica. Cases dependent upon the pressure or irritation of pelvic disease are called secondary sciatica.

**Symptoms.**—The onset of sciatica may be either sudden or gradual. The prominent symptom is pain along the course of the sciatic nerve, extending oftentimes into its ultimate branches. This pain is aggravated by pressure over the nerve, and by all movements which place any unusual tension on it. The character of the pain varies in different cases, or even in the same case from time to time. It may be dull or sharp, darting, tearing, burning, or boring. It is nearly always worse at night. It is quite frequently associated with a variety of morbid sensations, these including numbness and tingling in the area supplied by the sciatic nerve.

Motor symptoms are of two classes. One—and that most frequently encountered—is due to the increase of pain excited by any motion. To relieve tension on the inflamed nerve the patient walks with flexed knee. The second class is the result of weakness or actual paralysis of the muscles to which the sciatic nerve and its branches are distributed. These latter are usually limited to severe cases. Then the muscles are apt to be flabby, and even atrophied.

Trophic symptoms are sometimes observed. Herpes is very exceptional, but coldness and other evidences of enfeebled local circulation are not uncommon.

**Diagnosis.**—There are many conditions which have been diagnosed as sciatica, *e. g.*, hip-joint disease, disease of the spinal cord, and muscular pains in the legs, but it seems to me that even ordinary care should protect from such errors. The chief difficulty is found in the differentiation of primary from secondary cases. The persistence and agonizing severity of the pain is usually the first indication of some serious trouble hitherto unsuspected. Possibly the association of constitutional disturbance as high fever, may suggest the possibility of the presence of some other inflammatory condition. The recognition of the cause of a secondary sciatica can only be made by thorough physical examination of the pelvic organs through the rectum or vagina, or both, and by bimanual palpitation. A diagnosis of sciatic neuritis is only admissible when a careful survey of the totality of the symptoms enables us to exclude disease in all other situations.

**Prognosis.**—So far as ultimate cure is concerned, the prognosis of sciatica is favorable. Most cases run a very tedious course. The duration of the case, other things being equal, is proportionate to the severity of the symptoms. Some cases may last one or two years. Whether the obstinacy of the affection to remedial means is necessary or not is uncertain. The majority of cases occurring in men who must be up and about to earn their livelihood, probably explains why many are of such long duration.

**Treatment.**—The first element in the treatment of a case of sciatica is absolute rest. The more acute the case, the more rapid its onset, the



greater the necessity for such rest. When it is impossible to secure sufficient quiet by simple rest in bed, still more absolute quiet by the application of splints should be secured.

The splint used should be light in weight and of a kind capable of checking every motion at the hip and knee. The bandage keeping it in place should be of flannel, and extend from foot to groin, and applied with a moderate degree of pressure. Usually, two or three weeks of splint rest are required to make the treatment of an old case of sciatica successful. But even then it is advisable to inculcate care on the part of the patient, and keep him under observation for some little time longer. After removal of the splint, the bandage should be applied twice daily for some time. In cases not very severe the bandage alone is very efficient.

In all convalescent cases the greatest judgment should be exercised to avoid bringing pressure to bear on the diseased nerve, as the slight pressure from sitting on a hard chair may bring the trouble back with all its old severity.

Cases not yielding to other measures may be treated by dry cups, of which a number may be applied over the course of the sciatic nerve; or again by counter-irritation, as from a mustard plaster or the Pacquelin cautery.

Some obstinate cases of sciatica make surprisingly rapid recoveries on the discovery and removal of a simple cause which keeps up the trouble. As examples of this we have the numerous instances in which constipation has been the trouble, and free purgation has given entire and permanent relief.

Hot or cold applications are often of value, both as palliatives of the pain and as actual curative agents for the cure of the inflammation. Some cases tolerate heat, others cold. The use of cold is attended with considerable difficulty, owing to the care required in maintaining a uniformly low temperature. This may be secured by placing the limb on a suitably prepared tin box which has been filled with ice.

Of palliative remedies for the relief of the pain, the most important is *acetanilid*, which may be given in doses of from five to ten grains, cautiously repeated, and that too only after knowing the idiosyncrasies of the patient. Very severe cases may find their sole relief in morphia hypodermically. These, in my experience, are decidedly exceptional. As in all other painful affections, the danger of forming the morphia habit must be borne in mind.

*Electricity* should only be brought into requisition when the active inflammatory stage of the disease has subsided. Galvanism only is to be used. The current strength may vary from eight or ten to thirty or more milliamperes. The electrodes should be large flat ones. The positive pole should be placed over the sensitive points in the course of the

nerve, usually over the sacro-sciatic notch, and the negative over the lumbar spine. The current should be as smooth and as free from interruptions as possible. The sittings should be daily, and of from five to fifteen minutes' duration. Galvanism sometimes gives some wonderful results in the treatment of old sciaticas.

In acute cases, *gelsemium* and *aconite* are my favorite remedies. The former is applicable to cases presenting no special indications; the latter to cases arising from exposure to cold, and attended by prominent sensory symptoms, as tingling, etc.

*Rhus* is the best remedy for the subacute and chronic cases of rheumatic origin, especially when they have arisen from exposure to cold or dampness, or from overexertion. The characteristic *rhus* modality is generally present. I believe this remedy adapted to the majority of chronic cases. Some few rheumatic cases do well under the administration of salol, salicin, salicylic acid or the salicylates. It must be remembered that these are exceptional.

*Belladonna* should be employed in cases coming on suddenly, and characterized by the severity of the pains, and the high degree of inflammatory action.

*Colocynth* has been highly lauded in cases in which the pains are cramplike, but I have never used it. Jousset gives as an additional indication for it, sense of constriction around the haunch. Sometimes the pains are of a burning, boring character, and the paroxysms are followed by numbness.

*Turpentine*, in doses of from two to five drops, is advised by Hale, in cases dependent upon or associated with some irritation of the urinary organs.

*Gnaphalium* is a good remedy in sciatica. The attacks of pain alternate with periods of numbness.

*Kali hydriodicum* is the great remedy in syphilitic cases. It must be administered in large doses to secure the full result.

Some few cases resist all medicinal and hygienic measures. These may be treated by nerve stretching, and very successfully at times. So-called subcutaneous stretching of the nerve is occasionally efficient. The patient is thoroughly anæsthetized, after which, with the leg extended on the thigh, the latter is strongly flexed on the abdomen. This failing, the cutting operation should be tried.

Great claims have been made by Valentine Gibson for acupuncture of the nerve itself as a remedy for sciatica. He has treated one hundred cases by this method, fifty-six of which were cured, and but two were failures. If these results were obtained from the treatment of obstinate cases exclusively, they should be considered very good, but that they were of such a nature is extremely doubtful, as it is hardly likely that such a large number of the really bad cases should fall to the lot of any

one individual. The method advocated is worthy of trial when all other measures fail. It consists in puncturing the nerve with a spear-pointed needle. The patient can always tell when the nerve has been pierced by pain shooting down the leg. The needle should be withdrawn immediately after its introduction. Several punctures may be made at a sitting, making but the one cutaneous entrance. The operation may be repeated at intervals of two or three days. Sight must not be lost of the fact that these patients, as do all others recovering from surgical operations, obtain the effects of rest in bed.



# DISEASES OF THE BRAIN AND ITS MEMBRANES.

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## CEREBRAL PACHYMENINGITIS.

Pachymeningitis, or inflammation of the dura mater, may be divided into two varieties, the external and the internal. In the former the outer layer, or that which serves as the periosteal covering of the inner surface of the cranial bones, is involved. It is practically always secondary to traumatism or disease of the cranial bones. It occurs usually after fractures. More rarely, it complicates caries of the temporal bone in mastoid and middle ear disease, and caries of the ethmoid bone in long-standing ozenas. It has also been known to complicate erysipelas and syphilitic disease of the cranium.

**Symptomatology.**—The symptoms of pachymeningitis, though well marked, are by no means characteristic, being those common to many acute intracranial affections. Following traumatism, they set in usually within from twenty-four to seventy-two hours after the accident. Severe headache is prominent, the head usually being sensitive to touch. Then, too, we note delirium, fever, more or less stupor, and sometimes convulsions and paralysis. Suppuration very frequently takes place, and pus accumulates between the dura and the cranium.

**Treatment.**—This is strictly surgical. Trephining is imperative, and that, too, whether the fracture be a depressed one or not. There should be no delay in urging the importance of the operation. Cases secondary to syphilitic disease of the cranium must be treated by anti-syphilitic remedies, for which see the article treating of syphilis of the nervous system. But even here surgical interference is, by no means, contraindicated.

## PACHYMENINGITIS HÆMORRHAGICA.

This is a variety of pachymeningitis interna, and is believed to be of rare occurrence. Still, it has been asserted that as soon as the profession is made aware of its existence, it will be found far from uncommon. It consists of an inflammatory growth of connective tissue into which a hæmorrhage occurs. The nature of the trouble is at present a matter of considerable discussion. Virchow and Seguin advocate the primary inflammatory origin of the trouble, while Wigglesworth, Roberson and Huguenin believe it to be essentially of a hæmorrhagic

character, and propose that it should receive the name of subdural hæmatoma.

**Etiology.**—It is observed chiefly in males and in persons of advanced years, especially in those beyond the age of seventy. It may occur in infants, however. It very rarely exists as a primary affection, generally complicating certain forms of insanity, notably general paralysis of the insane and chronic alcoholism. It may follow certain of the acute diseases, as smallpox, rheumatism, typhus fever, relapsing fever and scurvy.

**Pathology and Morbid Anatomy.**—The lesions are nearly always bilateral. Between the dura mater and arachnoid are found numerous layers of a membranous tissue. Within these layers may form sacs, into which blood is effused. These clots undergo degeneration or become organized.

**Symptomatology.**—The symptomatology of this affection is very obscure. Some cases occur in which no phenomena whatever mark its onset. The symptoms that may suggest its existence are apoplectiform symptoms resulting from the small hæmorrhages which occur from time to time, optic neuritis, nystagmus, mental and physical weakness.

In children the occurrence of convulsions, twitchings and hemiplegia in conjunction with scorbutus or rickets, suggests a diagnosis of this disease.

**Diagnosis.**—The clinical picture not being at all characteristic, one is obliged to rely upon the previous history of the case. When the symptoms above enumerated occur in a patient suffering from general paralysis of the insane or chronic alcoholism, the provisional diagnosis of pachymeningitis hæmorrhagica is justifiable.

**Prognosis.**—This is very unfavorable.

**Treatment.**—This must be conducted entirely on theoretical considerations. As far as I know, there are no reports on the practical results. The hypothetical hæmorrhagic origin of the trouble calls for measures directed to the control of intracranial hæmorrhage, as elevation of the head and shoulders, rest and application of cold to the head. The inflammatory theory calls for the employment of anti-inflammatory measures.

### PURULENT INTERNAL PACHYMENINGITIS.

This is even more rarely observed than is pachymeningitis hæmorrhagica. It consists of an inflammation of the inner layer of the dura mater with suppuration. It has no distinctive symptoms.

## ACUTE CEREBRAL LEPTOMENINGITIS.

The term acute leptomeningitis is used to indicate an inflammation of the pia mater, of a non-tubercular character. It is generally spoken of as meningitis, and sometimes as brain fever. It may be either simple or purulent.

**Etiology.**—The most frequent cause of acute leptomeningitis is traumatism. There seems to be no definite relation between the severity and nature of the injury, and the character of the resulting inflammation. It is not necessary by any means that there be an external wound of the scalp. In some cases, especially when the patient was intoxicated at the time of its reception, the injury has failed of recognition, the resulting meningitis being taken for delirium tremens. Meningitis is very liable to occur secondarily to quite a variety of acute diseases. Of these, pneumonia is probably the most frequent. Other diseases worthy of mention in this connection are smallpox, typhoid fever, inflammatory rheumatism, whooping cough, measles and scarlet fever. Meningitis may arise from extension of inflammation from neighboring structures. It may follow the operation of enucleation of the eyeball, or disease of the middle ear or of the cranial bones, whether syphilitic or otherwise. It may very rarely occur as a primary affection as the result of syphilis. Other constitutional diseases in which it may originate are gout and Bright's disease.

In some cases it has been alleged to be metastatic in origin. Improved and more thorough methods of investigation throw great doubt on such a theory, for almost invariably the disease can be traced to one of the other causes above enumerated.

**Pathology and Morbid Anatomy.**—The inflammation may involve any or all portions of the pia mater. Usually in those cases dependent upon pneumonia or endocarditis, the pathological condition is limited to the convexity of the brain. When it has arisen from extension of disease from the middle ear, it is apt to be limited to the corresponding side of the brain, and is then not infrequently complicated by abscess or thrombosis of the cerebral sinuses. The inflamed pia mater is found thickened and opaque. There is likewise an injected condition of the vessels of that membrane. The subarachnoidal fluid is increased in quantity and is turbid. Sometimes this increase is sufficiently great to cause flattening of the convolutions and empty surface capillaries by the pressure thus exerted. The pathological findings are not always limited to the membranes, for in some cases the brain tissue itself beneath the inflamed parts is softened. In cases which go



on to suppuration a layer of creamy pus is found covering the pia mater. Exceptionally, it may be subdural.

**Symptomatology.**—The symptoms of acute meningitis are of very rapid onset. Usually the disease is ushered in by a rigor and headache. These symptoms are quickly—so quickly as to be almost simultaneously—followed by fever and delirium. These four symptoms—fever, headache, vomiting and delirium—constitute the main features of meningitis, and call for extended mention. The headache is always severe, and is subject to paroxysmal exacerbations, during which the patient is obliged to cry out because of his sufferings. Fever, though a very constant phenomenon, is high only exceptionally, rarely going above 103° F. The vomiting may present almost any grade of severity. It may occur but once or twice, or it may be nearly constant. It comes without any evidence of gastric disorder, and generally disappears late in the disease. The delirium presents no features to distinguish it from the same symptom, as it occurs during the course of typhoid fever and other acute diseases.

Associated with the symptoms already described are certain phenomena which, if present, serve to increase the probabilities of a diagnosis of meningitis. Vertigo is one of the earliest of these to appear. The special senses are hyperæsthetic; light and noise cause discomfort. The face is flushed; the eyes are bright and injected; and the pupils are contracted. Sometimes epileptiform convulsions occur and are repeated frequently. Local or general rigidities may appear; especially characteristic is a rigidity of the muscles of the neck. Ischæmia of the retina or neuro-retinitis is sometimes present.

At the end of the stage of irritation, stupor gradually increasing to coma comes on. The pupils hitherto responding sluggishly to light, now become dilated. The face becomes pale, and the extremities cold and bathed in a cool sweat. The epileptiform convulsions, if present before, are continued, or may now show themselves for the first time. The patient may even die during the continuance of one of them. The evacuations become involuntary.

The duration of an attack of meningitis will vary with the intensity and extent of the inflammation. Those cases in which the membranes covering the entire brain are affected rarely last more than three days. Less severe cases may last much longer.

**Diagnosis.**—The diagnosis of meningitis can never be made with absolute certainty. The most that we can say is that meningitis is probably present. The four systems above mentioned—fever, headache, vomiting, and delirium—are very strongly suggestive of it. Especially do they become so when their appearance is associated with any of the causes of the disease as already outlined. Especial care must be taken that a symptomatic delirium in the course of an acute febrile disease is not misunderstood.

**Prognosis.**—Acute meningitis is always a very serious disease. The possibility of recovery from it has even been questioned. I think that nearly every one will in the course of his professional career observe cases in which permanent brain injury, optic nerve atrophy, or other nervous phenomena, have continued after attacks of disease presenting all the classical symptoms of meningitis. In view of the uncertainty of a diagnosis in any event, care must always be observed never to give an absolutely unfavorable prognosis, no matter how bad the outlook.

**Treatment.**—The general treatment of simple or suppurative meningitis does not differ in any particular from that outlined for the tubercular variety. It is well to shave the head. Douches of cold water are of some value in the comatose stage. Lumps of ice should be administered to allay vomiting.

Surgery offers considerable hope in the cases depending upon traumatism and middle ear disease. Still it must not be resorted to until the indications for operation are plain and it is reasonably certain that the patient cannot recover under careful nursing and medical treatment.

In ear troubles it is probably best to direct all surgical interference to the ear directly. As promptly as possible, the mastoid and antrum must be opened, cleared of all septic matter, and thorough drainage affected. Trephining, excepting as it furthers these ends, is unwise.

In cases arising from traumatism, *arnica* is the remedy *par excellence* unless there are clear indications for others. It should be given as a prophylactic from the very beginning of accidents liable to produce meningeal inflammation, as there is considerable evidence of its value. In the convalescent stage of the disease, when mental dulness or apathy is present, it should also be administered.

*Aconite* is adapted to the early stages of the disease, that is, on the first appearance of inflammatory symptoms. Especially is it called for in cases arising from exposure to the sun.

*Belladonna* is the remedy in cases presenting violent symptoms. The delirium and headache are intense. The face is greatly congested. The patient's manner is quick and hasty. When symptoms of stupor, paralysis, etc., appear, it is no longer indicated.

Then *bryonia* in particular is to be thought of, as probably the stage of effusion has been reached. Jahr praised this remedy, saying that it is indicated when the delirium is of a mild type, and the pains in the head are of a severe shooting, tearing character. The face is congested a dark red. There are sensorial depression, constant chewing motion of the mouth, shooting pains worse from any motion. Fever may or may not be high.

When the convulsive phenomena are prominent, then *cuprum* should be given. The face is pale and the lips blue; delirium is marked; and there are violent movements of the eyeballs.

*Lachnanthes* has been recommended for the rigidity of the neck.

In syphilitic cases no remedy is equal to *mercury* or *iodide of potassium*. The latter is called for in the chronic cases especially. I have seen a most remarkable recovery occur in a case of meningitis, occurring about one year after infection, under the influence of mercurial inunction after mercury by the mouth and iodide of potassium had signally failed.

*Veratrum viride* has been suggested by Hale as a valuable remedy to alternate with belladonna in all cases in which that remedy is indicated.

Other remedies to be studied as applicable to meningitis are *glonoin*, *digitalis*, *helleborus*, *gelsemium*, *solanum nigrum*, *œnanthe crocata* and *zincum*.

### CHRONIC CEREBRAL LEPTOMENINGITIS.

Chronic leptomeningitis is so nearly always as to amount to practically always a secondary affection. It occurs in association with tumors, disease of the bloodvessels, suppurative disease of the ear, alcoholic poisoning, syphilis and tuberculosis.

Its symptoms present no characteristic features, consisting of headache, vertigo, vomiting and impairment of memory and other mental faculties. The recognition of the disease must then be largely conjectural, and depend mainly upon the knowledge that one of the causes of the condition exists, and the ability to exclude all other diseases liable to produce similar symptoms.

At the autopsy it is recognized by thickening and opacity of the membranes, and sometimes by adhesions between the different layers of the meninges, or of these to the brain substance.

**Treatment** only offers any hope when syphilis is the cause of the trouble, in which case iodide of potassium must be given in large doses, as recommended in the article on syphilis of the nervous system.

### LEPTOMENINGITIS INFANTUM.

Leptomeningitis infantum is the name given to an inflammation of the pia mater about the base of the brain not dependent upon tubercular infection. It has also been called "*hydrocephalus sine tuberculis*" and "*occlusive meningitis*." The latter title was received because the foramen of Magendie is often closed by the inflammation, leading to a retention of a ventricular effusion.

The **Symptoms** differ in but few particulars from those of tubercular meningitis. The most prominent symptom is the rigidity of the neck. It is liable to occur very shortly after birth in debilitated children.

The **Prognosis** is highly unfavorable, though recovery may ensue even after exudation has taken place. It is very apt, under such circumstances, to lead to chronic hydrocephalus, imbecility, moral depravity and other evidences of impaired intellect.

The **Treatment** is the same as that recommended for tubercular meningitis. (See page 346.)



## HYDROCEPHALUS.

Hydrocephalus is an accumulation of serum within the cranial cavity. The effusion is generally limited to the cavities of the ventricles, in which case it is called internal hydrocephalus. In other cases it is subdural, and then is described as external hydrocephalus. The existence of the latter variety has been denied by good authority. In former years it was customary to speak of acute and chronic hydrocephalus. Inasmuch as the former cases are all inflammatory in origin, this nomenclature has been abandoned, and that class described as tubercular meningitis.

**Etiology.**—The vast majority of cases of internal hydrocephalus are congenital. Their origin has been ascribed at times to inherited defects, as syphilis, inebriety, etc. As to the former, I feel satisfied that a sufficient number of cases of hydrocephalus are the offspring of syphilitic parents to establish a relation between these affections. Many cases are associated with rachitis, an important point to bear in mind, for, as will hereafter be stated, great difficulty in differentiating hydrocephalus from the latter sometimes obtains. Injuries to the child *in utero*, uterine disease, violent emotional influences acting on the mother, have each been assigned as a cause. Not infrequently the disease exists *in utero*, thus constituting a cause of dystocia.

**Pathology and Morbid Anatomy.**—There are current two prominent views respecting the origin of hydrocephalus, one ascribing it to inflammatory troubles, and the other to mechanical causes arising from tumors and other organic diseases closing one or more of the foramina leading from the ventricles. Less prominent, because urged by fewer authors, is the theory that the accumulation arises from deficient resistance of the cranial bones. The latter are almost always very thin in hydrocephalus, and it is believed are unable to resist by normal pressure, the ordinary accumulation of serum within the ventricles. Thus they allow of dilatation, instead of promoting absorption. The proper view to hold, I believe, is that each of these influences may become etiological factors. The effused serum does not always present the same characteristics, ranging in specific gravity from 1001 to 1009; sometimes it presents the features of ordinary subarachnoid fluid, while in others it partakes of the inflammatory type. In quantity it may be found from a few ounces upwards, the largest quantity obtainable in any case within my knowledge being twenty-seven pints. The condition of the brain itself is dependent upon the extent of the effusion. When this

is great, the ventricles are correspondingly distended, and the surrounding brain structure forms a thin sac-like wall. The convolutions on the surface are obscured or even obliterated. It may be impossible to distinguish the gray from the white matter. The basal ganglia become indistinguishable. Different structures are absent or destroyed by the exorbitant intracranial pressure. The cranial bones themselves are thinned, at times so much so as to show the brain structure within as soon as the scalp has been raised. It is not at all unusual for them to be quite translucent. The ependyma of the ventricles sometimes shows a granular aspect; at others it is perfectly normal. The lateral ventricles are generally affected to a greater extent than either the third or the fourth. Sometimes it is possible to demonstrate obliteration of one of the foramina, the foramen of Monroe, Magendie, etc.

**Symptomatology.**—Not infrequently, as already stated, the trouble makes itself known at birth by reason of the enlarged head. In other cases, it comes on within the first weeks or months of life. Usually the enlargement of the head is the first thing to direct the attention of the attendants to the state of affairs; exceptionally, the onset of the disease may be marked by general emaciation, irritability, convulsions, etc. The increase in the size of the head varies greatly in individual cases, it reaching 107.6 cm. in a case mentioned by Gowers. It is the rule for the increase to be uniform in all directions. Exceptionally, the antero-posterior diameter is most affected. The face does not keep pace with the growth of the cranium, this disproportion giving the little sufferer a peculiar appearance. The sutures are widely separated, and the membranes tense and bulging. The scalp itself is thin; the hair is sparse. The superficial veins are prominent.

In mild cases, and very exceptionally even in them, the cranial enlargement may be associated with no other symptoms whatever. In the majority of instances, other phenomena are noteworthy. Owing to the weight of the head, the child is unable to hold it erect, especially when the muscles are weak, as they usually are. The orbital plate of the frontal bone is pushed downward by the exudation. This condition is said to occasion the appearance of the eyes peculiar to hydrocephalic children. A considerable portion of the lower half of the iris is covered by the lower lid, while quite a margin of the sclerotic above is visible. It is claimed by some that the superior muscles are weak, and that the deviation of the eyes arises from this cause. Rolling of the eyes, ordinary nystagmus, and different varieties of strabismus, or disturbance of vision may be present. The latter is occasioned by atrophy of the optic nerve, which may come on from pressure of the effusion on the optic chiasm, from stretching of the nerves or secondary to optic neuritis. The former is by all odds the most frequent condition.

The muscles of the body generally are weak. This is especially true

of those of the legs. The mental condition of the patient is more or less impaired. Sometimes, it amounts to simple backwardness, and at others to complete imbecility.

Epileptiform convulsions sometimes attend the progress of the disease.

**Diagnosis.**—The chief difficulty lies in the differentiation of hydrocephalus and rachitis. In the latter, the head is large but square, the forehead is bulging, and there are evidences of rachitis elsewhere. Then, too, the increase in the size of the head is comparatively slight in the latter affection. Hydrocephalus is impossible of recognition in the absence of the cranial enlargement. In the investigation of this point, certain measurements should always be made. They are: (1) The circumference of the head at the level of the glabella and occipital protuberance; (2) from one mastoid process over the vertex to the other; and (3) from the root of the nose over the vertex to the occipital protuberance.

**Prognosis.**—Most cases end fatally within a few months, or at the most a few years. Certain cases not very far advanced may be stayed in their course, the morbid tendency seeming to wear itself out. Cases in which life has been preserved for years have been reported. Mental improvement is scarcely to be expected.

In some few cases a spontaneous cure has been effected by the discharge of the serum through the nose or through the scalp.

**Treatment.**—Very little positively reliable can be stated concerning the treatment of hydrocephalus. One cannot in a disease like the one under consideration speak authoritatively without an unusually large experience. This no one seems to have had. Considerable can be offered in the way of suggestions. One measure that has gained considerable reputation, and has been followed by some favorable results, is strapping of the head by adhesive plasters. A better procedure is the application of a band of elastic webbing, three inches in width, about the head at the level of the forehead. Care must be taken that the pressure is not so great as to produce redness of the parts or leave the imprint of the material on the skin. Sloughing may be produced by too tight pressure.

A measure that has received but little attention is the application of solar heat. Several physicians have reported their experiences with it. Locatelli reports one case cured; Nicita treated three cases successfully; while Somma obtained good results in five cases. The method consists in exposing the child's occiput to the direct rays of the sun for twenty minutes each day, gradually increasing the duration of the seance until the limit of thirty or forty minutes is reached. It is believed that the local sweating acts to remove a portion of the effusion, while the thermic irritation aids nutrition. Rodionoff reported a case in which the patient



was neglected by the mother after being well cared for until two years of age. The little one was more or less constantly in the sun. A spontaneous recovery ensued within a year.

Surgical measures, tapping of the ventricles, incision and drainage, have been recommended; but I cannot see that the reports of cases thus treated give us very much hope. Still, I should not hesitate to recommend one of these procedures as a last resource, for Unverricht, Phocas, and, it is presumed, others, have made considerable claim of the value of the procedure, based on personal experience. Only one or two ounces of fluid should be evacuated at a time. Quinke has recommended tapping of the spinal canal between the third and fourth lumbar vertebra and draining off one or two ounces of fluid. This method seems to possess but little danger.

The medicinal treatment of hydrocephalus must never descend to the level of routinism. The administration of remedies for the removal of effusions, as *apis*, *apocynum*, etc., will meet with disappointing results. Certain deep-acting remedies, like *calcareo carb.* and *phos.*, *sulphur*, *silicea*, *baryta carb.*, *lycopodium*, *mercurius*, *kali hydriodicum*, *psorinum*, *arsenicum* and *tuberculinum*, should be studied. Burnett reports a cure made with *tuberculinum*, prescribed because of the patient's inherited diathesis. The indications favoring one or the other of these remedies are those evidencing constitutional peculiarities, and not the symptoms of hydrocephalus *per se*. The majority of the latter are purely mechanical, and can have but little weight in the selection of a remedy acting dynamically.

### EXTERNAL HYDROCEPHALUS.

External hydrocephalus is nearly always of inflammatory origin. It is acquired; rarely congenital. It is frequently met with in the aged as secondary to atrophy of the brain structure. It may be encountered in quite a variety of cases attended by diminution in the size of the brain. The symptoms attending the condition are not such as to make its recognition at all certain during life.

### CEDEMA OF THE BRAIN.

The clinical picture presented by œdema of the brain is not sufficiently characteristic to dignify it by separate mention in this work. Owing, however, to the fact that cases occasionally occur in which autopsy reveals no other condition present than this, I feel that completeness requires its mention. Œdema of the brain may occur secondarily to disease of the heart or kidneys, and to anæmia. Secondary to the former, it is usually the result of obstructive congestion from mitral stenosis; in association with Bright's disease it has, on several occasions, produced unilateral convulsions simulating in every way those generally

regarded as diagnostic of focal brain disease. I reported one such case a number of years ago, although the full significance of the phenomena was not at that time understood. Anæmia of the brain may excite a high degree of œdema. The autopsy in such cases sometimes reveals extensive softening of the brain substance, said by some to be due to maceration of brain tissues by the fluid, and by others to this plus post-mortem changes.

Sometimes the œdema accompanies atrophy of the brain substance, as in cases of imbecility and idiocy and chronic inflammation of the membranes. The fluid is found between the membranes and within the substance of the brain itself. On section of the latter, the clear fluid is observed to well up.

As already stated, no definite diagnostic symptoms are presented. The condition, therefore, is not recognizable during life.

## CEREBRAL HYPERÆMIA AND ANÆMIA.

### CEREBRAL HYPERÆMIA.

By cerebral hyperæmia is meant an excessive quantity of blood in the capillary vessels of the brain. Concerning its actual existence both as an actual clinical entity and as an accompaniment of other morbid conditions the greatest differences of opinion prevail. One author requires page after page to describe the symptoms significant of the condition and the post-mortem appearances in fatal cases; while another contents himself in using up the entire space devoted to cerebral hyperæmia to endeavoring to prove its non existence. As usual when two such extreme views exist, the truth is probably to be found between them. Space will not permit me to advance the arguments bearing pro and con on this question. Let it be sufficient to state that there are unquestionably cases of cerebral hyperæmia both primary and secondary; and also that this condition is diagnosed altogether more frequently than attention to scientific accuracy should permit. Many of the cases thus named are undoubtedly instances of hypochondriasis or neurasthenia or one of the numerous organic cerebral affections.

**Etiology.**—Cerebral hyperæmia occurs normally under great excitement or unusual mental exertion. It is natural to believe that the persistence of these external agencies to an unnatural extent should transfer a normal temporary change into a permanent pathological one. Cardiac disease, especially overaction and hypertrophy of the heart, may be attended with excessive cerebral blood supply. Especially is the latter trouble operative when combined with aortic regurgitation. External cold by exciting contraction of the cutaneous vessels may produce cerebral congestion. Certain poisons, notably alcohol, nitrite of amyl, nitroglycerin, etc., may be a cause. It is also believed to occur as one of the conditions present in sunstroke. Suppression of menstruation may produce it; and there is strong evidence to show that it is not infrequently the cause of much of the suffering incident to the climacteric.

Passive congestion of the brain is always the result of mechanical causes. Anything which impairs the return flow of blood from that organ may produce it. Thus cardiac diseases which cause an overfilling of the venous system, violent attacks of coughing, great muscular effort with closed glottis, playing of wind instruments, tumors obstructing the venous flow from the head, are all to be considered in this connection.

**Pathology and Morbid Anatomy.**—Cerebral hyperæmia may be



either active or passive. In either condition there is excessive quantity of blood in the capillaries; but in the former it is arterial, while in the latter it is venous and dependent upon a hindrance to the flow through the veins.

Many have been the post-mortem appearances assigned by authors as evidence of the existence of cerebral congestion during life. All of these may now be regarded as unreliable; it is even questioned, with good reason, if there can be any post-mortem evidence of this condition, whether active or passive. It is known that active congestion on the surface of the body, *e. g.*, the areola about a pimple, disappears at death. In the case of passive congestion there is undoubtedly dilatation of the capillaries; but the significance of this fact is counterbalanced by the very frequent occurrences of post-mortem congestions from hypostasis.

Cerebral congestion is sometimes secondary to neurasthenia, which impairs the functions of the vaso-motor nerves.

**Symptoms.**—These are more marked as the onset is sudden. A cerebral hyperæmia of quite high grade may exist without symptoms, if its onset has been gradual. The symptoms themselves attending the condition are not very characteristic. In active congestion we look for paroxysms of headache, delirium, and sometimes fever, preceded or attended by throbbing of the bloodvessels of the head and neck, and associated with redness of the face. Mental excitement, tinnitus aurium, vertigo and insomnia, are other clinical features of the disease.

Passive congestion is characterized usually by dull frontal headache, confused feeling in the head or sense of fulness. This is the form of headache that may be produced by the violent fits of coughing. The face and eye are deeply congested.

Transient apoplecticiform seizures with short-lasting hemiplegia have been attributed to simple congestion; but this explanation of their cause is exceedingly unlikely, notwithstanding it is the only one we can reasonably offer in the present state of cerebral pathology.

**Diagnosis.**—This is a very difficult matter. Care should be taken, however, that cases having the already described symptoms, but associated with evidence of focal lesion, are not diagnosed as simple hyperæmia. Before offering cerebral hyperæmia as the diagnosis of a given case, I should want to make sure by every method of examination at my command, that all other conditions can be excluded.

**Prognosis.**—As to individual attacks, the outlook is very favorable indeed in the vast majority of cases. Occasionally the symptoms may present a high degree of intensity, and justly give rise to some alarm. The character of the primary disorder is always an important point for consideration. The so-called congestive attacks in locomotor ataxia, disseminated sclerosis and general paralysis of the insane are not infrequently fatal.

**Treatment.**—All cases call for a careful study, and removal of the cause. Without this, success is impossible. A quiet mode of living, both physical and mental rest, and a moderate nutritious diet form the most important elements in the general management of the patient.

Remedies must be selected not only by the symptoms of the hyperæmia, but also, and this is of greater importance, by the primary condition. This opens up a wide therapeutic field which the limits of the present article will not permit me to review.

*Aconite* is called for in active congestion resulting from cold, violent emotions, or excited action of the heart. The arterial tension is high; the general surface of the body is cold.

Ever since homœopathy became a system of medicine, *belladonna* has been a popular remedy in cerebral congestion, especially when headache is a prominent symptom. The latter is of a throbbing character, is aggravated by the recumbent posture, and is associated with marked throbbing of the arteries of the head and neck. There is great feeling of heat about the head, while the extremities are cold. Either drowsiness or sleeplessness may be associated symptoms, or these conditions may even alternate. Hughes praises this remedy as the best for the cerebral congestion of childhood.

*Glonoin* is called for in cases arising from sunstroke and menstrual suppression. The attack is sudden, and of the active variety. There is throbbing pain in any or every part of the head. The face is deep red.

With *aurum* the cerebral hyperæmia is intensified, if not actually caused by cardiac hypertrophy. There is a sensation as of rush of blood to the head, aggravated by mental exertion. There are also feeling of fulness in the head, tinnitus aurium, sore and bruised sensation in the head, sparks or flashes of light before the eyes.

*Veratrum viride* is indicated in febrile conditions in which cerebral hyperæmia is a prominent feature.

*Gelsemium* and *ferrum phos.* are indicated in passive congestions.

*Nux vomica* is indicated rather by the associated conditions and causes. It is called for in persons of sedentary habit and addicted to use of alcohol and animal food in excess.

*Sulphur* is indicated in cerebral congestion when attended by tinnitus aurium and redness of the face, the latter symptom being aggravated in the open air, and better in the warm room.

Electricity will prove an invaluable adjuvant in chronic cases. The galvanic current should be passed from the forehead to the back of the neck, applying the positive electrode in the former situation. Rumpf has recommended the application of the faradic brush to the head. This influences the intracranial circulation favorably by reflex action from the skin.

## CEREBRAL ANÆMIA.

Local cerebral anæmia, such as arises from the obstruction of one of the arteries of the brain, will be fully described in the sections devoted to embolism and thrombosis of the vessels. The present article will, therefore, be limited to the consideration of general cerebral anæmia.

**Etiology.**—The most frequently observed cause of cerebral anæmia is an impairment in the quantity or quality of the blood, as in the different varieties of anæmia (chlorosis, pernicious anæmia, leukæmia, etc.) and following hæmorrhage. Sometimes the cause resides in the condition of the heart (that organ being weak), or in obstruction to the passage of blood headward by an intrathoracic tumor. Sudden cerebral anæmia may be produced by sudden dilatation of the intestinal vessels. The best example of the trouble arising from this cause is found in the syncope and even death which follows the rapid removal of intra-abdominal pressure after paracentesis abdominis. It may likewise arise as the result of Bright's disease, obliterating endarteritis, disorders of digestion and nutrition, and the encroachment of large tumors, hæmorrhages, etc., upon the cranial cavity.

**Pathology and Morbid Anatomy.**—The brain is always found extremely pale, and devoid of the puncta vasculosa. Effusion of serum is found to have taken place, as if to occupy the space lost by the deficient vascular supply. The vessels, with the exception of the large ones of the dura, are full.

**Symptoms.**—Cerebral anæmia may be divided into the acute and the chronic varieties, each of which presents its individual symptomatology. The acute variety follows hæmorrhage, or is caused by sudden psychical influences. Its symptoms may be summed up in the one word syncope. There occurs in rapid succession obscuration of the senses, as dimness of vision, impairment of hearing, tinnitus aurium, vertigo and loss of consciousness. The general surface of the body is pale and cold; respiration is labored, first accelerated and then slow. If the case is a severe one, as after large hæmorrhage, coma, convulsions and death supervene.

In the milder cases, which usually result from psychical causes or systemic weakness, the attack is generally preceded by mental incoherence, oppression of the chest, followed by coldness of the surface with clammy perspiration, tinnitus, nausea and even vomiting, and loss of consciousness.

*Chronic cerebral anæmia* is, as already intimated, usually associated with general impairment of the quantity or quality of the blood. The condition, in these cases, has been summed up as one of irritable weakness. The patient has fainting spells from slight causes, as already described. Mental effort is difficult; undue excitement follows slight



influences. The patient has also vertigo, headache, tinnitus aurium and even delirium. Insomnia is often a troublesome symptom. General muscular weakness and hallucinations of the special senses are frequent phenomena. There is marked amelioration of all symptoms from the recumbent posture. Cardiac palpitation, especially upon exertion, is often complained of.

**Prognosis.**—This must largely depend upon the character of the primary disorder. If organic disease of the various viscera may be excluded, the favorable prospects are enhanced. Cases dependent upon pernicious anæmia offer a poor outlook indeed. It is important always that a cure be accomplished as soon as possible, for long-continued malnutrition of the brain cannot help but exert an unfavorable influence on its development for all time to come.

**Treatment.**—In attacks of syncope, the patient should at once be placed on the floor or in bed with the head low. Cold water should be dashed in the face, and friction of the surface of the body practised. When severe hæmorrhage has been the cause of the trouble, then infusion of salt solution, or auto-transfusion performed either by elevating both lower extremities, or in direr necessity driving the blood from the arms and legs by compression with an Esmarch or ordinary roller bandage, becomes a positive necessity.

In all cases, absolute rest in bed must be enforced. The fewer changes in posture the patient makes, the better it will be. The diet must be of the most nourishing character possible, and likewise readily digestible.

Internal medication is largely influenced by the primary disorder. Remedies like *ferrum*, *arsenic*, *phosphorus*, *nux vomica*, etc., will be most frequently available.

## APOPLEXY.

The term apoplexy has been applied to quite a variety of pathological conditions of the brain, the characteristic symptom of which is sudden onset with loss of consciousness. The name was derived from the Greek word ἀποπλήσσω, which means "I strike down." Inasmuch as most of the cases thus denominated resulted from rupture of a blood-vessel and the consequent escape of blood into the cerebral substance, the terms apoplexy and intracranial hæmorrhage became almost synonymous. So common did this error in nomenclature become that all hæmorrhages into the substance of the different viscera became commonly known as apoplexies; thus we hear of apoplexy of the lungs, apoplexy of the retina, apoplexy of the spinal cord, etc., in all of which conditions sudden striking down unconscious is characteristic by its absence. Of late years a better tendency to call things by their right names prevails. Instead of designating a case as one of apoplexy, it is now customary to call it by the name of the pathological condition producing the symptoms.

The disturbances giving rise to apoplectic seizures all find their origin in the circulation; they are as follows: (1) Intracranial hæmorrhage; (2) embolism of a cerebral bloodvessel; (3) thrombosis of a cerebral vessel. The most frequently observed of these is intracranial hæmorrhage. This, from its importance, will now be considered *in extenso*.

### INTRACRANIAL HÆMORRHAGE.

Intracranial hæmorrhage may take place either into the substance of the brain itself or about the meninges. When the latter, it is nearly always traumatic in origin. This variety will be the subject of separate consideration.

**Etiology.**—A subsequent section of this article will endeavor to demonstrate that cerebral hæmorrhage can only occur, aside from traumatism, in the presence of disease of the vascular structures of the brain. This being the case, it is evident that the predisposing causes of the condition under consideration must be those active in the production of vascular degeneration. Of these causes, age stands forth prominently. It is decidedly exceptional for cerebral hæmorrhage to take place prior to the fortieth year of age. Indeed, I must express, as my individual opinion, that its occurrence in patients not yet of middle life is highly improbable, if renal disease or syphilis can be excluded. The excep-

tional is, however, not impossible; accordingly we may meet with rare instances of cerebral hæmorrhage attacking young adults, even as early as the twentieth year. With advancing years the number of cases increases, the maximum, in point of actual numbers, being probably reached at about the sixtieth year; though the relative proportion of cases grows greater with each additional year. Age is made an influential factor by reason of the arterial degeneration which comes with advancing years.

Renal disease is unquestionably a very prominent factor in the production of cerebral hæmorrhage. Not only is it active in causing arterial disease, but the increased intra-vascular tension and the accompanying cardiac hypertrophy favor rupture because of the great strain brought to bear on the arterial walls. Especially active in this respect is chronic interstitial nephritis. Authors do not, in my opinion, assign to the different varieties of Bright's disease their proper importance as etiological factors in the condition under study. Fagge is an exception, he bearing testimony to the regularity with which renal disease is found in the autopsies.

Men are more frequently affected than women, doubtless by reason of habits and occupation. The proportion has been variously given, some making it as great as ten to one, while others say that men are more frequently seized in the proportion of two to one.

Excessive indulgence in alcohol over a term of years is a powerful predisposing cause. Especially will this poison be productive of mischief in those accustomed to drink moderately large quantities with regularity, though never becoming intoxicated. Not only is there a direct effect by the alcohol on the vessels themselves, but likewise another intensifying the first by reason of its damaging influence on the kidneys and other important viscera.

Certain acute diseases predispose to attacks by producing serious blood changes. These are typhoid fever, diphtheria, scarlatina, purpura, scurvy, pernicious anæmia, and leukæmia.

Heredity once held a high position in popular esteem as a cause of so-called paralytic strokes, much importance having been attached in the past to the so-called apoplectic habit. Patients of this class were said to be those of plethoric appearance, short thick neck, and stout. Strümpell, of all authors, appears to be the only one who still holds to this idea, which has some basis in fact, in that gouty people usually present the apoplectic habit above referred to. The inheritance of the gouty diathesis is doubtless a powerful predisposing cause.

Occupations which expose persons to radiating heat, especially when great bodily exertion is required, and those which necessitate uncomfortable postures, must be regarded as predisposing to cerebral hæmorrhage.

The exciting causes are those agencies which temporarily increase



the intracranial blood pressure. Powerful emotions, falls, unusual exertion, violent fits of coughing, great straining at stool, or partaking of a heavy meal, may bring on an attack. It is worthy of note that the majority of cases occur during cold weather, especially when sudden barometric changes take place. Patients are apt to remain in a crowded and hot room for some time and get overheated. Thus the vessels of the surface become relaxed and distended. Going out into the cold air excites strong contraction of the previously dilated vessels, and increases the intravascular tension. The arteries being diseased and unable to bear with the strain, cerebral hæmorrhage ensues.

A large number of cases occur during sleep; why, has not been satisfactorily explained. It has been said that the contraction of the bloodvessels during this state and the dependent position of the head afford an all-sufficient reason.

**Pathology.**—By far the most frequently observed pathological cause of cerebral hæmorrhage is the so-called miliary aneurisms. These were first described and their importance urged by Charcot and Bouchard, who demonstrated their presence in every one of seventy-seven consecutive cases. They rarely occur in subjects of less than forty years of age, and are of more frequent occurrence with advancing years. They find their starting point in a diffuse periarteritis. Thickening of the lymph sheaths and wasting of the muscular coat takes place, and the aneurisms are formed. They vary in size from 0.2 mm. to 1 mm. in diameter, and in number in individual cases from two or three to several hundred. They are most frequently observed about the basal ganglia, the corpora striata and the optic thalami, and in decreasing order, the centrum ovale, the cortex, the pons and the cerebellum. While the rupture of these aneurisms is oftentimes occasioned by a sudden or a persistent increase in the intravascular blood pressure, it must be remembered that such increased pressure is by no means necessary to that accident.

A diffuse periarteritis without the formation of the miliary aneurisms may lead to the vascular rupture.

Atheroma plays a subordinate part in the production of cerebral hæmorrhage. This form of degeneration is limited to the larger vessels, the walls of which it renders decidedly less elastic than in health. It is the smaller arterial branches that rupture. It is generally held that the loss of elasticity by the larger vessels causes the full force of the heart-strokes to be expended on the terminal vessels, a strain they are unable to withstand.

Syphilitic disease of the arteries likewise leads to weakening of their walls. It has not yet been satisfactorily demonstrated that syphilis can cause miliary aneurisms.

Investigation shows that in the majority of cases of atheroma miliary aneurisms exist. Still one may readily be present without the other.

It has been held by a few that the rupture of the aneurisms is favored by a preliminary softening of the cerebral substance, which acts somewhat as a support. Whether or not the softening favors the rupture, it is undoubted after the investigations in the post-mortem rooms at Guy's Hospital, as reported by Fagge, that these aneurisms favor a lowered state of cerebral nutrition, and therefore a softening. This softening must be productive of impaired brain function.

The effused blood forms a clot which may vary in different cases from an almost infinitesimal size to a bulk of several ounces. It produces symptoms either by pressure on adjacent nerve fibres, or else by rupturing the same. Usually but one extravasation is found; still they may be multiple. When recent, the clot consists of a dark loose coagulum. Usually it is mixed somewhat with disintegrated brain tissue. After a few days it shrinks in size, and at the same time becomes darker in color, becoming brown and finally reddish yellow. It is very apt to excite inflammation in the surrounding structures, and this results in the proliferation of connective tissue and the formation of a limiting membrane or cyst wall with degenerated clot as its contents. The latter show a tendency to be absorbed while the cyst wall itself contracts. In exceptional instances, complete absorption takes place, and only a cicatrix remains. Occasionally a cyst with clear fluid contents is the final result.

Hæmorrhage into the ventricles is nearly always the result of the extension of an extravasation into the caudate nucleus ploughing its way through the brain substance. Rarely does it occur from rupture of the vessels of the choroid plexus. Generally but one ventricle is affected; still the hæmorrhage may extend itself throughout the ventricular cavities by way of the connecting foramina.

In the majority of cases of hæmorrhage into the internal capsule (by all odds the most frequent site of cerebral hæmorrhage), there follows a secondary degeneration of the motor tracts below. It is this phenomena to which the late rigidity of hemiplegia is due.

**Symptoms.**—The frequency with which premonitory symptoms are experienced in apoplectic seizures must remain uncertain. In the vast majority of cases no history of such is obtainable from the patient or bystanders. Yet it is inconceivable that the seizure should come on with the remarkable suddenness usually described. The hæmorrhage is almost always from a small arterial branch, and must take an interval of time to become operative in symptom production. The attack always occurs unexpectedly, and bystanders, unless they be unusually accurate observers, cannot help but overlook premonitory symptoms unless they are especially obtrusive. Occasionally the seizure is preceded for several days by certain indefinite symptoms arising from cerebral disturbance. These include sensory disturbance in the extremities of one side, formication, numbness and tingling, feeling of heaviness in the limbs, and

peripheral pains. Sometimes there is headache or feeling of fulness in the head, or there may be choreiform movements of the parts about to be paralyzed (pre-hemiplegic chorea). Two explanations for the existence of premonitory symptoms so long before the seizures have been offered. One is that they are the result of the impaired nutrition of the brain consequent upon the vascular degeneration, and the other, that they depend either upon minute extravasations, or a slowly increasing hæmorrhage.

The seizure itself is in a vast majority of cases announced by sudden unconsciousness. This may reach its maximum intensity at once, and be so profound that the patient is absolutely incapable of being affected by any external impression, however strong (apoplectic foudroyant). In other instances disturbance of cerebration is evident; the patient appears bewildered, loses his way in well-known places, and this increases until stupor supervenes, and this finally becomes more or less profound.

The complete stupor is characterized by loud snoring respiration. The pulse is slow, full and strong. The face is deeply congested. Both tendon and superficial reflexes are unobtainable. The pupils have usually lost their power to react to light. They vary greatly in size in individual cases. The duration of the stupor is by no means constant. Sometimes consciousness is regained within an hour, within a few minutes even. In aggravated cases, it lasts for days; indeed the patient may never come out of it. Improvement in the stuporous condition is evidenced by restlessness, or by susceptibility of the patient to external impressions which formerly had exerted no effect.

In some few cases of cerebral hemiplegia, consciousness remains undisturbed. This is known as the simple mode of onset. The patient experiences a slight weakness which compels him to sit down, and this rapidly increases until complete hemiplegia appears. Not infrequently the attack takes place during sleep, and the patient wakes in the morning hemiplegic.

There is a rare form of cerebral hæmorrhage in which the development of the symptoms is not abrupt. In the course of a few hours paralysis develops, and this is followed by a slowly increasing coma. It is termed *ingravescent apoplexy*.

Hemiplegia comes on with the stupor; but owing to the patient's condition is not manifested until consciousness is regained. Exception to this latter statement may be found in cases in which the loss of consciousness is not profound. Then examination of the sound side shows a certain amount of resistance to passive movement of the extremities, while the paralyzed side is perfectly lifeless. Usually all of one side, face, arm and leg, is paralyzed. Exceptionally the face escapes, or the face is paralyzed on one side, and the arm and leg on the other. This latter distribution of the paralysis is ordinarily spoken of as *alternate*



hemiplegia, or, more correctly, crossed hemiplegia. It is diagnostic of a lesion in the upper half of the pons on the side corresponding to the affected side of the face. During the comatose stage, the facial paralysis may be betrayed by the drooping of one corner of the mouth, or by exaggerated flapping of one cheek. It is a significant fact that muscles which are accustomed to act bilaterally, *e. g.*, the respiratory muscles of the chest, are never severely affected. Muscles more or less associated in their functions with those of the opposite side recover rapidly. Thus the paralysis of the face soon disappears. The leg regains its function long before the arm. The large muscles of the shoulder and hip recover before the smaller ones of the foot and hand. Patients do not observe any trouble with the so-called sound side. Accurate investigation, however, shows that even this in incurable cases has lost one-third of its wonted strength.

The character of the facial paralysis is worthy of more extended remark. The lower muscles of the face only are affected. The orbicularis palpebrarum and the forehead muscles escape. Voluntary movements are more affected than are involuntary or emotional. Thus difficulty is experienced in attempting to make a grimace, while emotional movements as those of laughing and crying are but slightly impaired.

The hypoglossal nerve is frequently involved in the paralysis. This is first shown by difficulty in articulation. Owing to paralysis of the genio-glossus, the tongue is not protruded straight, but deviates to the paralyzed side. The paralysis of the tongue may be permanent. In most instances, fortunately, it recovers quickly.

The temperature generally displays marked departure from the normal. Bournonville, who has given this subject considerable study, claims that there is an initial fall to even as low as  $96^{\circ}$ . After a period of twelve or more hours, there is a rise, which may reach in fatal cases  $108^{\circ}$  or  $109^{\circ}$  or more. This observer has made the thermometer an important instrument of prognosis. I do not think that the initial fall described by Bournonville can be confirmed in every case. I am satisfied that sometimes the rise in temperature begins at once. It is certain, however, that cases attended by either marked rise or fall present very poor chances for recovery. After a few days there may be another accession of fever due to inflammatory disturbance about the extravasation or to pulmonary complications or bedsores.

The temperature of the paralyzed limbs is usually at first from one to two degrees higher than their fellows, probably because of vaso-motor paralysis.

In severe cases a most interesting phenomenon, known as conjugate deviation of the head and eyes, is observed. This consists of a turning of the head and eyes to one side; the patient appears to look over one shoulder, as it were. The deviation is usually towards the side of the

lesion in the brain. Attempts to retain the head in the median line are futile, for it at once returns to its abnormal position. This is always a serious symptom. When rigidity or convulsion develops, then the deviation is towards the convulsed side of the body and away from the side of the lesion in the brain. It may occur in conjunction with hæmorrhage into the pons, in which case the deviation is the reverse of that above described, being away from the side of the lesion in paralysis and towards it in case of rigidity or convulsion.

Vomiting is sometimes a symptom, more especially when the hæmorrhage invades the cerebellum.

Convulsions sometimes announce the commencement of the attack. This mode of commencement is especially characteristic of hæmorrhage into the cortex. It may occur, however, in conjunction with lesions elsewhere.

Sensory disturbances are variable. When present, anæsthesia is not profound as a rule. When marked, it is characteristic of a lesion in the posterior portion of the posterior limb of the internal capsule. Usually the sensory symptoms are limited to a sense of numbness and formication. Sensibility to touch is more affected than is that to pain. Loss of muscular sense is sometimes experienced.

The special senses are occasionally affected. Hemianæsthesia may be observed. Smell and taste are rarely involved. Hearing is almost always disturbed, though never destroyed.

During the comatose stage micturition and defecation may be involuntary. On the other hand, there may be retention of urine, which, as the bladder becomes distended, gives place to dribbling. Ofttimes the urine is found markedly albuminous. Dana claims that this latter symptom may occur independently of any renal disease; but of the correctness of this assertion I entertain grave doubts. I believe that where the albuminuria is at all great, or where it is more than ephemeral, structural changes in the kidneys exist. Case after case will be met with in which, with the improvement in the nervous phenomena, the renal condition likewise changes for the better; but in all such coming under my notice I have demonstrated organic disease of the kidneys.

In certain severe cases, fortunately rare, sloughing bedsores develop. These generally go from bad to worse very rapidly.

For the first few days after the attack the deep reflexes are absent on the paralyzed side; then a change takes place, the tendon reflexes show marked exaggeration, especially on the paralyzed side.

Sometimes, at the end of the third or fourth day, improvement ceases, the temperature unexpectedly rises, and dyspnoea, cyanosis, and other evidences of lung disease, appear. The patient usually dies, and, at the autopsy, extreme congestion of the lungs is found, especially marked

on the paralyzed side. It has been claimed, and with good reason, that this congestive pneumonia is a trophic lesion of the brain disease.

**Chronic Stage.**—By this stage of the disease is meant that in which all active symptoms have passed away and the patient has the disabling phenomena remaining. Of these, the hemiplegia will be found to be the most prominent. This will have improved considerably over its initial severity. Especially will the improvement be noted in the leg. Instead of the paralyzed parts being flaccid as before, they are now rigid, this latter phenomena being spoken of as the late rigidity of hemiplegia. In the arms, rigidity of the flexor muscles predominates; in the legs, it is the extensors that are more affected. The muscles are rarely atrophied to any great extent, only, as a rule, to the degree one would expect from the enforced idleness. Their electrical reactions are normal.

The patient's mental condition is nearly always impaired somewhat. Usually, this is in the direction of increased emotional impressionability. He is more irritable than formerly; or he is given to sudden outbursts of temper. Exceptionally, the psychical condition exhibits a progressive tendency to dementia.

A rare trophic phenomena in hemiplegia is arthropathy. The joints on the paralyzed side are all affected. The trouble consists of a multiple synovitis with plastic exudation, with a tendency to formation of ankyloses. The affected parts are hot, painful, and very sensitive to the touch.

A study of the associated movements in hemiplegia is of practical interest. They consist of movements of the paralyzed extremity, when the patient moves the corresponding unaffected one. Sometimes, the reverse condition occurs, the patient making movements of the sound limbs on trying to move the paralyzed ones.

Other peculiarities in the movements of the affected parts are found in the choreic movements, the so-called post-hemiplegic chorea. This complication consists of disorderly movements, aggravated by emotional influences, and every attempt to execute a voluntary movement. They cease entirely during sleep. They are analogous to the athetoid movements, which are practically continuous during waking moments. These post-hemiplegic motor disorders are especially frequent after the hemiplegias of infancy and childhood.

**Diagnosis.**—The diagnosis of the seat of lesion, in cases of intracranial hæmorrhage, is not without great scientific interest, though it may be devoid of practical therapeutic value. I cannot but feel it incumbent upon me to give a review of the subject.

Hæmorrhage into the cortex is recognized by the appearance of convulsions as the initial symptom. These spasms are of local commencement, may or may not be associated with unconsciousness, and are followed by paralysis of the previously convulsed parts.



In hæmorrhage into one of the crura, there is paralysis of the face, arm and leg, on the one side, and of the motor-oculi nerve, on the other. Hæmorrhage is rarely limited to this locality, however.

Hæmorrhage into the pons is frequently ushered in by convulsions. Usually, these are general. Occasionally, they are limited to the legs. Generally, the paralysis affects both sides; but, when the lesion is a small one, it may consist of hemiplegia only. When the lesion is in the lower half of the pons, this hemiplegia is of the alternate type, the face on one side and the arm and leg on the other being affected. The pupils are strongly contracted; respiration is apt to be embarrassed.

Hæmorrhage into the medulla is rapidly fatal. Even a very small hæmorrhage into this locality is attended by rapidly fatal results.

Ventricular hæmorrhage is indicated by the occurrence of a second apoplectic stroke shortly after the first, together with the extension of the hemiplegia from one to both sides of the body.

There are several conditions which may be confounded with cerebral hæmorrhage, if but little of the patient's personal history is known, and this is apt to be the case when the attack comes on with the victim away from home.

*Syncope* may thus be mistaken; but here the loss of consciousness is the result of failure of the circulation. The face is, therefore, pale; the pulse is weak, often hardly discernible; respiration is sighing; the reflexes are preserved.

The separation of *uræmic coma* from cerebral hæmorrhage is always difficult, sometimes clinically impossible. The uræmic coma is not likely to be of sudden onset; it is not infrequently preceded by convulsions. The coma is not apt to be so profound as that of apoplexy, and this enables us to investigate the movements of the extremities. Suppression of urine is, of course, a valuable point. Albuminuria does not possess much significance, for diseased kidneys are frequently found in hæmorrhagic cases.

The most frequent mistake is made in confusing apoplectic patients with "drunks." The odor of whiskey on the breath counts for but little, for so many patients are seized after an evening spent in carousing. In alcoholic coma, the patient is rarely, if ever, as profoundly unconscious as in cerebral hæmorrhage. There is almost always some restlessness, by which the absence of hemiplegia can be recognized. There is no conjugate deviation of the head and eyes. Convulsions, if present, partake of the character of struggling, instead of possessing the epileptiform type.

*Dementia paralytica, disseminated sclerosis* and certain other organic diseases of the central nervous system are associated with attacks of apoplectiform character. These can only be recognized by a knowledge of the history of the patient. Bournonville has claimed, but with insufficient reason, that the apoplectiform differ from the true apoplectic seizures in the absence of the initial fall of temperature.

The differential diagnosis of cerebral hæmorrhage, embolism, and thrombosis will be considered in the articles devoted to the latter affections.

**Prognosis.**—No matter how mild an attack of apoplexy from cerebral hæmorrhage may be, it is always a serious matter; serious not only as to the immediate results, but also as to the future of the patient, should he make an apparently perfect recovery from the attack. The rupture of a cerebral vessel indicates a serious condition of the vascular system. There are certain data which aid us to a certain extent in forecasting the chances of recovery. The more profound the coma, the worse is the outlook, both as to life and as to permanent hemiplegia. A great initial drop in the temperature is unfavorable, as is also a high fever. Very, very few cases indeed, recover to such an extent as to leave no objective evidence of the seizure. Exceptional cases in which the primary symptoms were severe and long-lasting, have made comparatively prompt recoveries.

**Treatment.**—Preventive measures do not offer very much hope of success. The disease finds its origin in a vascular degeneration, and this can only be retarded in its onward progress by careful attention to all hygienic measures, and internal medication directed to current conditions. Unfortunately it is seldom that the first recognition of vascular degeneration comes before the accident has happened.

During the attack itself, the first indication for treatment is absolute rest; and by absolute rest I mean rest in every possible particular. The importance of this point cannot be overestimated. It has been claimed by some that the damage done by an apoplectic extravasation is done in an instant. This is a mistake, for after the rupture of the vessel the symptoms very frequently increase in intensity for an hour or more. Very often patients are encouraged by their friends to keep moving in order to throw the symptoms off. This is most pernicious advice. So complete should be the rest that endeavors to arouse the patient by calling to him should be desisted from. Passive movements of the body must be positively forbidden.

Attention to the patient's posture is important. The head should be high. If stertor is present, the patient should be placed upon his paralyzed side. Lying on the side stops the stertorous breathing, which is not only distressing to the friends, but also increases the arterial tension. It is important to have the paralyzed side undermost, because the movements of the healthy side are then free. There is also the theoretical reason that the affected side of the brain will be uppermost, and thus away from the influence of gravity in perpetuating the hæmorrhage.

The clothing must be loosened, and the head so placed as to avoid flexion of the neck, thus doing away with all obstruction to the return flow of blood from the brain.

In getting the patient to bed, every possible care as to the above details must be followed. If there is the slightest difficulty in undressing him, there should be no hesitation whatever as to ripping open the seams of his clothing.

When collapse is not present, the application of ice to the head has a very beneficial effect, as it serves to excite contraction of the cerebral vessels. When collapse is present, mustard plasters to the nape of the neck have been recommended by Gowers as of value in inducing reflex contraction of the arteries. It is well, likewise, to wrap the legs in cloths wrung out in hot mustard water; a cupful to a pail of water.

The lancet as a means of reducing arterial tension has been very properly abandoned by all intelligent physicians in the treatment of apoplexy. We may, however, employ the device first formally suggested by Dr. Dawbarn, of New York, to "bleed the patient into his veins," so to speak. As soon as possible the physician should cut off the return circulation from the lower extremities. This is done by the application of an Esmarch bandage, a tourniquet, or Spanish windlass, to one or more of the extremities, and as near the trunk as possible. The apparatus should be made sufficiently tight to obstruct the return flow through the veins pretty thoroughly, but not so much as to interfere with the arterial flow. Experimental evidence shows pretty clearly that this procedure lowers arterial tension, and favors the cessation of internal hæmorrhages. The pressure should be kept up for about an hour. Then the blood should be permitted to enter the general circulation very slowly. The only objection to Dawbarn's suggestion is that of possible danger in the hands of those who are unskilled and who lack judgment. Persons of that kind should never undertake the treatment of a human being, no matter how mild his ills; consequently the objection fails to have weight.

Sometimes it is well to get the mechanical effect of a violent purge. For this purpose one or two drops of croton oil should be put on the tongue.

There are cases, many of them, indeed, in which all our best-directed efforts fail of relief, and deep and long-lasting coma supervenes. These must be treated on general principles. The bedding requires the most careful attention, owing to the danger from bedsores; in extreme cases the water-bed is desirable, if not absolutely necessary. Extreme cleanliness must be enjoined.

When the extremities are cold, hot water bottles should be used. They should be applied with the greatest care, however. Owing to the patient's helpless condition the liability to produce burns is great, and a burn in a hemiplegic patient is a serious matter, for local nutrition is poor and the danger of sloughing correspondingly great.



The diet *must* be the lightest possible. Liquid articles, as milk and broths, should be the sole articles permitted during the acute stage.

In all cases stimulation by alcohol is bad practice.

Should there be a very high temperature (105° F. or higher), I should certainly have recourse to the *cold* pack. I have used it in other brain affections with high temperature with excellent results, though as yet not in apoplexy.

Antiseptic cleansing of the mouth and pharynx is an important hygienic measure.

Retention of urine calls for catheterization; especially is this an important measure when retention of urine is followed by dribbling.

As to medicines, if in the beginning there is an excited condition of the circulation, *aconite* should be administered. It will almost certainly have a beneficial effect.

If the cerebral congestion be a prominent symptom, *belladonna* should be thought of, especially with the characteristic circulatory disturbances of that remedy.

*Glonoin* I would advise in cases in which the arterial tension is high and there is coexisting kidney disease. One drop of the first centesimal dilution should be given three times daily. As the administration of the drug is continued, it may be given at shorter intervals until the patient is taking it every two or three hours.

*Opium* should be thought of in cases characterized by marked venous congestion. The profoundness of the stupor is not an indication for the drug, because that is dependent upon the severity of the case and therefore upon mechanical causes only. For this opium, or in fact any other drug, is powerless.

*Arnica* is the drug that should be administered after the acute symptoms have subsided to promote absorption of the effused blood.

For the subsequent paralysis *causticum* has done more good in my hands than any other remedy. It is of course impossible to say how much of the improvement in these cases is due to drug, and how much to time, which is certainly an essential element in their cure and improvement.

*Sulphur* and *baryta carb.*, the latter especially in old people, have likewise been recommended as remedies that will promote the absorption of the clot.

Attention to the kidneys is always an important matter. Whenever there is any albuminuria or excess of uric acid, I advise the use of Londonderry or Buffalo lithia water, preferably the former carbonated.

When the subject of cerebral surgery was first broached it was thought that a possible remedy for apoplectic extravasations had been announced. Unfortunately, this is not so, for it takes but a little thought to see at once that surgical interference in the vast majority of cases is

worse than useless, probably harmful. When the symptoms are such as to point without question to a sub- or extra-dural hæmorrhage, much may be accomplished, providing, of course, that the cerebral arteries are not too far advanced in their degeneration. When, on the other hand, the hæmorrhage is in the corpora striata, the effused blood cannot be liberated without seriously damaging important brain fibres.

Electricity is often proposed as a remedy in apoplectic paralysis. There is great danger that the pressure brought to bear by the family may lead to its use either too early in the case or in entirely unsuitable cases. I would advise that the patient be permitted to enjoy rest without electrical interference. In the course of a month or so the application of galvanism to the head may prove useful by promoting absorption of the clot. Galvanization of the contracted muscles and faradism of their opponents have been recommended when the stage of late rigidity has come on. I have had very little success with these measures, and have ceased encouraging patients to resort to them. I have decidedly more confidence in the applications to the head.

Horsley has recommended ligation of the common carotid on the side of the lesion as a means of stopping the internal hæmorrhage. There can be no doubt, if we are to judge from experimental evidence, of the efficacy of this procedure, but the operation is of such a severe character, and requires so many precautions to render it safe, that by the time it has been performed the hæmorrhage has ceased spontaneously. Carotid compression is probably as efficient.

Something can be done in the late stages of post-apoplectic paralyses by properly directed gymnastics of the paralyzed parts. The aim should be to call the healthy side of the brain into play to help the injured one. This may be done by directing similar movements to be performed on both sides of the body simultaneously. It is astonishing how much better are the movements of the paralyzed extremity when thus performed than when the limb is made to move by itself. This is a field that has not been thoroughly developed, but I think it one of some promise.

The apoplectic patient is in every instance a disabled one. Though he should be so fortunate as to recover without a semblance of paralysis, he is still largely incapacitated for his former labors. Ever afterwards he should live, as far as possible, quietly and abstemiously, enjoying the balance of his life as best he may.

## EMBOLISM OF THE CEREBRAL ARTERIES.

Obstruction of the cerebral arteries by either embolism or thrombosis is also generally known as acute softening of the brain, because of the pathological changes produced in that organ by this accident.

By embolism is meant the plugging of one of the arteries by a piece of fibrin or other foreign material detached from some distant portion of the vascular system.

**Etiology.**—Some authorities teach that embolism occurs more frequently in men than in women. Fagge and Osler, on the other hand, show from the statistics at their disposal that this is hardly correct, and that this difficulty occurs with equal frequency in the two sexes.

It occurs oftenest between the ages of twenty and fifty, though no period of life is exempt. It is especially rare in children.

Emotional influences, by producing sudden disturbance in cardiac action, may precipitate an attack of cerebral embolism by causing the detachment of a particle of fibrin or vegetation from one of the valves.

The important causes of embolism, however, are endocarditis, endarteritis and other morbid conditions which lead to the formation of fibrinous deposits on the valves, and thrombosis in different vessels. When the former, it is the valves of the left side of the heart, especially the mitral, that are affected. The endocardial disease is usually a fresh warty inflammation on sclerosed valves. Rarely does it complicate the endocarditis of acute inflammatory rheumatism and the acute fevers. Sometimes the embolus comes from the aorta, the result of atheroma or aneurism of that vessel. In still other cases it originates in the lungs, and is carried through the pulmonary veins to the left side of the heart. In the latter case the embolus may consist of septic material, and, in addition to exciting the usual alterations in the brain attendant upon embolism, may produce abscess or minute foci of suppuration.

**Morbid Anatomy and Pathology.**—Any cerebral artery may be the seat of the trouble. It is, however, more frequently met with in the middle cerebral, the left it is said with far greater frequency than the right; this because the course of the blood current to the left middle cerebral artery is more direct. Fagge denies this generally accepted statement, and shows by the statistics of Guy's Hospital that the left artery is but slightly more frequently obstructed than the right. He explains the misconceptions of others concerning this point on the supposition that they have drawn their conclusions from the report of cases scattered throughout medical literature. The unusual interest attached



to the subject of aphasia has led to the report of cases associated with this symptom, and this necessarily brings greater prominence to embolism of the left middle cerebral artery of which aphasia is a by no means unusual symptom.

The obstructing plug may consist of decolorized fibrin or a warty vegetation from one of the valves, a portion of a thrombus, or a calcareous mass from an atheromatous aorta. It is usually arrested in a vessel at a point where it divides or its lumen is narrowed by the giving off of an important branch. Following the obstruction there ensues a coagulation of blood beyond the embolus to the minutest distal branches, while on the proximal side, coagulation extends only to the nearest branch. In some cases the plug may be broken up into numerous small pieces, and become dislodged before coagulation can occur.

The obstructing plug is not always discoverable at the autopsy. This is probably because it has undergone absorption. Fagge offers the further suggestion that the point of obstruction may have been at the bifurcation of the carotid in the neck or at one of its bends within the petrous bone.

The influence exerted by the obstruction on the brain tissue supplied by the affected artery is of the greatest importance. If the embolus is so situated as to block vessels possessing anastomotic connections, the collateral circulation is soon established, and the nutrition of the area of distribution is but little affected, permanently at least. If, on the other hand, terminal branches are involved, necrosis of the brain within the affected vascular area follows. From the above it will be inferred what is really the case, that the arteries of the brain may be divided into two sets, one terminal, possessing no anastomoses with other vessels, and the others having thorough connections of that character. The former set supplies the deep basal ganglia, and the latter the cerebral cortex.

The first effect of the arterial obstruction is the production of anæmia of the area to which the involved artery is distributed. Sometimes this is associated with small capillary ruptures and the consequent minute hæmorrhages. In the course of twenty-four or forty-eight hours changes in the brain tissue commence. Serum is effused and infiltrates the surrounding tissues; the brain substance breaks down and local softening ensues. This softening has been described as of three varieties, red, yellow and white. This division is, however, without any clinical importance, as it depends entirely upon the amount of the blood or its coloring matter within the softened area, or else the changes that have taken place in it. Red softening is noted with especial frequency in the cortex, where the vascular supply is free. Yellow softening is but a late stage of red softening, in which degenerative changes in the effused blood have taken place. White softening is of the same color as the normal white substance of the brain. The consistence of softening is very vari-

able. It may be almost liquid in one case, and scarcely less than the normal brain consistence in another. In all cases there is no sharp line of demarcation between the degenerated and the normal areas, the softened substance gradually merging into that of normal firmness.

The softened portion is almost always surrounded by a zone in which inflammatory changes have taken place.

The ultimate changes in the lesion are three-fold: The softened area may remain unchanged for years; there may appear an abundant formation of connective tissue about the softening, and this becomes firm and dense, the encapsulated matters become absorbed, and a cicatrix results; and lastly, the solid particles within the connective tissue formation are absorbed, leaving only the fluid contents, and a cystic tumor remains.

**Symptoms.**—There are no premonitory symptoms in cerebral embolism. This is readily understood, when it is remembered that there is no disease in the brain or its appendages prior to the seizure; the embolus originating from without, its presence in the brain is purely a matter of accident. The onset of the symptoms, therefore, is sudden, with or without unconsciousness. Their general character is that already described in the article on cerebral hæmorrhage. There are the general or transient symptoms ushering in the attack and the permanent or localizing symptoms. The unconsciousness in embolism is usually of short duration. Epileptiform convulsions usher in the seizure with far greater frequency than in hæmorrhage. They may either be general, or else Jacksonian in type and limited strictly to the parts subsequently found to be paralyzed. Aphasia, a disorder of speech to be described hereafter, is of frequent occurrence because the left middle cerebral artery is so frequently the seat of the embolus.

Delirium sometimes replaces the unconsciousness as an initial symptom. When inflammatory reaction appears, this symptom is apt to increase.

The mind is less likely to be permanently affected than it is in hæmorrhage.

**Diagnosis.**—The differential diagnosis of cerebral embolism and cerebral hæmorrhage at times forms a most difficult if not, indeed, a nearly impossible clinical problem. In a general way, it must be borne in mind that the most important data for differentiation are found in etiological evidence. A history of endocarditis, inflammatory rheumatism, aneurism, organic valvular disease, etc., points strongly to embolism, especially when the patient has not yet reached an age at which arterial degeneration becomes a probable factor. The relation between the degrees of severity in the initial symptoms and the extent of the permanent or local symptoms gives considerable aid. In this connection it is well to impress upon the reader the fact that hæmorrhage is of

the two the more likely to produce severe initial phenomena. A small lesion with loss of consciousness is apt to be a hæmorrhage; a large lesion with preservation of consciousness favors embolism. A short period of unconsciousness favors embolism. In hæmorrhage, the disturbances in temperature are more marked than in embolism. The pulse in hæmorrhage is apt to be of high tension and bounding.

**Diagnosis of the Vessel Affected.**—The following are effects produced by occlusion of several of the cerebral vessels:

**VERTEBRAL ARTERY.** Acute bulbar paralysis. This may be unilateral and associated with hemiplegia.

**BASILAR ARTERY.** Sudden death from involvement of the respiratory centres. In complete occlusion, bilateral paralysis and bulbar symptoms. Hyperpyrexia. Rapidly fatal issue.

**POSTERIOR CEREBRAL ARTERY.** Softening may exist without symptoms. In some cases, hemianopsia from involvement of the branch going to the cuneus; hemianæsthesia from blocking of the branch to the posterior part of the internal capsule.

**INTERNAL CAROTID.** Hemiplegia, generally temporary, though sometimes permanent, may follow ligation of this vessel. When obstructed within the skull, the condition is far more dangerous, hemiplegia, coma and death being the usual result.

**MIDDLE CEREBRAL ARTERY.** Hemiplegia, and when the left side is affected, aphasia.

**ANTERIOR CEREBRAL ARTERY.** Dulness of intellect.

**Prognosis.**—This depends largely upon the vessel affected, as already indicated. The patient rarely dies during the comatose stage, a fatal result, when it occurs, being delayed until five or six weeks after the seizure. When there is to be a complete recovery from the paralysis, and other focal symptoms, it will be within the first few weeks.

**The Treatment** differs in but few particulars from that recommended under the head of cerebral hæmorrhage. Electricity, as an aid to the stimulation of cerebral nutrition, may be employed early, but a diagnosis must be clearly made out, as early electrical treatment in hemiplegia from hæmorrhage is not desirable.



## INTRACRANIAL ANEURISM.

Under this head will be considered aneurismal dilatations of the larger cerebral arteries only. This is not an unusual condition, as appears at first thought. The only statistics bearing on this point, those of Osler and Pitt, show that it has been found in twelve out of 800, and eighteen out of 9,000 autopsies respectively.

**Etiology.**—Intracranial aneurisms occur more frequently in males than in females. They may occur at any period of life from ten to sixty years of age, being of about the same degree of frequency in each decade. Injuries and syphilis, though undoubted causes, do not seem to be as important in the production of intracranial as of aneurisms elsewhere. In the case of injury as the starting point of the trouble, traumatic inflammatory changes in the intracranial structures follow, and these excite disease in the walls of the vessel, and dilatation ensues. Primary degeneration of the vessels is only an exceptional cause, and that in the second half of life. By far the most frequent cause is embolism, hence it will be unusual to find an intracranial aneurism independent of the existence of endocarditis or other well-known causes of embolism. In these cases the embolus only partially occludes the lumen of the vessel, and excites a local inflammation of the vascular wall, with subsequent thinning.

**Pathology and Morbid Anatomy.**—Intracranial aneurisms range in size from the size of a pea to that of a walnut. The middle cerebral arteries are the most frequently involved, because they are the favorite site for the lodgment of emboli. Next in frequency comes the basilar. Usually, the tumor is of the sacculated variety, communicating with the artery by a small opening. Very frequently the trouble gives no indications of its presence during life, until it ruptures and the patient dies suddenly in coma. In such cases it sometimes happens that the existence of the aneurism is discovered at the autopsy by the most careful search only.

**Symptoms.**—Sometimes there is, as already intimated, complete absence of all symptoms. In other cases, there may be the usual phenomena attendant upon small cerebral tumors. Of these headache is the most frequently observed. Usually, this partakes of the characteristics of organic headache, being constant; it may, however, be paroxysmal. Its relation to the seat of the lesion is governed by the same principles that apply to headache in all other organic brain diseases. When the aneurism is of the basilar artery, then the headache is almost

always occipital. Vertigo is a very constant symptom. Convulsions are rarely observed, notwithstanding the frequency with which the middle meningeal artery is involved. Owing to the involvement of the vessels of the brain, the cranial nerves are often pressed upon; consequently cranial nerve palsies constitute important diagnostic phenomena. Optic neuritis is rarely observed.

The following are some of the symptoms observed in connection with aneurism of several of the intracranial arteries:

**INTERNAL CAROTID ARTERY.** Aneurism of this vessel exerts pressure upon the optic nerve on one side, and of the nerves in the wall of the cavernous sinus on the other. Vision on that side is accordingly impaired, and there is associated with it paralysis of the third nerve. Ptosis is usually the first paralytic symptom, but all of the eye-muscles may suffer subsequently. There may be loss of common sensibility of the eyeball and of the sense of smell. When the aneurism is a large one, it may cause hemiplegia by compression of the crus.

**ANEURISM OF THE ANTERIOR CEREBRAL** produces about the same symptoms as the foregoing, with the exception that the third nerve usually escapes.

When the **MIDDLE CEREBRAL** is involved, paralyzes of cranial nerves are absent. Convulsions and hemiplegia may be present. Aphasia is an important symptom when the left artery is affected.

In **ANEURISM OF THE BASILAR ARTERY** the symptoms depend upon disturbance of the pons and the nerves finding origin thereabouts. Any or all of the cranial nerves from the third to the eleventh inclusive may suffer, though those from the fifth to the tenth inclusive are most frequently affected. Vertigo is very severe. Headache is occipital.

**Diagnosis.**—It is almost impossible to diagnose intracranial aneurism with certainty, excepting in those rare instances in which a murmur perceived on auscultation of the skull exists. This, when it occurs, is almost always in conjunction with aneurism of the internal carotid. It may even happen that the murmur is perceptible to the patient. An aneurism may be suspected with good reason, however, when added to the symptoms of a small tumor, as above specified, there are prominent causes of aneurism present to explain the trouble. The presence of arterial degeneration, or endocarditis, would thus favor aneurism. Syphilis, though an undoubted cause of the condition, would possess but little diagnostic value, because it is far more frequently the cause of syphiloma or chronic meningitis. The therapeutic test furnishes a valuable, though not infallible, guide. In the case of the latter condition, the iodide of potassium should effect a prompt improvement; this it will only do exceptionally in the case of aneurism. Another diagnostic guide is the recognition of the fact that the tumor is in the line of one of the intracranial vessels.

**Prognosis.**—The duration of intracranial aneurisms is very uncertain, because, in their early stages, they very rarely produce any symptoms whatever; consequently their time of origin always remains unknown. It is believed, however, that they may last all the way from five weeks to as many years. The prognosis is usually very unfavorable. Some few cases recover spontaneously, as the result of coagulation of the tumor's contents. Nearly all cases sooner or later rupture, causing sudden death. Aneurism of the internal carotid offers better prospects than any other variety, simply because it is so situated as to be amenable to surgical treatment.

**Treatment.**—This is the same as for aneurism elsewhere. Aneurism of the internal carotid calls for ligation of the common carotid. When the basilar artery is affected, ligature of both vertebrals has been suggested.

The medicinal treatment for the cure of aneurism has but little to commend it from the standpoint of results. *Baryta carb.* seems to enjoy a greater reputation than any other remedy, especially in elderly people with degenerated vessels. *Ferrum phos.*, *iodine* and *iodide of potassium* may be tried. The latter remedy should usually be given in small doses of the crude drug, that is from five to ten grains well diluted, three times daily.



## THROMBOSIS OF THE CEREBRAL SINUSES AND VEINS.

**Etiology.**—Thrombosis of the cerebral sinuses occurs either as a primary or as a secondary disorder. As the former it is met with especially in children suffering from marasmus, diarrhœa, or other exhausting diseases, and in adults during the closing hours of certain diathetic and exhausting conditions, as cancer, tuberculosis, etc. It is far more common, however, as a secondary affection, complicating long-standing suppurating disease of the middle ear, septic wounds of the head, especially in conjunction with fracture of the skull and inflammation of the diploë, erysipelas of the scalp and face, infective ulceration of the nasal fossæ, infective cellulitis of the orbit, periostitis of the jaws from dental caries, retro-pharyngeal abscess and anthrax.

Primary and secondary thrombosis have received also the names of marasmic or marantic and inflammatory or infective thrombosis respectively.

**Pathology and Morbid Anatomy.**—The peculiar anatomical conditions of the cerebral sinuses have much to do with the production of their thrombosis. They are large tubes of triangular form, with rigid walls, and with lumen occasionally obstructed by fibrous bands or trabeculæ. Then, too, the great longitudinal sinus is so situated as to put the flow of blood through it to serious disadvantage. In all of them any cause weakening the heart's action, retards the circulation through them. Exhausting diarrhœa deprives the blood of its watery constituents, thereby increasing the coagulability of that fluid. At the same time the total bulk of blood is diminished, and the sinuses cannot by reason of their rigid walls adapt themselves to the lessened quantity. Consequently their circulation is slowed. Marantic thrombosis is especially liable to occur in the longitudinal sinus, though it may appear elsewhere, even in the cerebral veins. In the latter case it often gives rise to symptoms of a localized lesion, as Jacksonian epilepsy, monoplegia, etc. The clot in marantic thrombosis is dense, in layers and not adherent to the walls of the vessel. Its tendency is towards organization, not disintegration. Sometimes it becomes tunnelled, and thus the circulation is re-established. It is believed that many of the cases of infantile hemiplegia with subsequent cerebral atrophy and sclerosis are caused by venous or sinus thrombosis.

Infective thrombosis, as a rule, affects the sinus nearest the site of the initial lesion. The walls of the vessel first show softening and

thickening, followed by coagulation of the blood within. This coagulum adheres closely to the walls of the vessel, and, owing to its infective origin, rapidly undergoes disintegration. The pus from the infected wall mixes with the disorganized clot, forming a greenish-brown fluid. This is rich in micro-organisms of highly infective character and which may readily produce widespread mischief by being carried into the general circulation. They may be spread through the circulation directly by being carried by the tributary veins, or through the but partially obstructed lumen of the sinus itself; or again, the infective matters may spread through the walls of the sinus, infecting adjacent tissues, and through them the general system. Very often, indeed, the neighboring structures undergo suppuration, and abscess results. The effect of the diseased sinus on the contiguous bone is exceedingly interesting. A marked discoloration with roughening and erosion of the bone is often observed.

Systemic infection from sinus thrombosis usually manifests itself in the lungs, though the liver and kidneys also may suffer.

**Symptomatology.**—The symptoms of sinus thrombosis possess the widest possible range; in fact, it cannot be said to have a well-defined symptomatology. In some cases all evidences of cerebral lesion are absent during life, and the trouble is not even suspected until revealed at the autopsy. In still other instances, the symptoms are so obscured by those of the primary disease as to escape observation. Such general symptoms as headache, dulness of intellect, gradually deepening coma, vomiting and convulsions, are not uncommon. The headache is observed early, and is very severe. It may occupy any portion of the head, yet at the same time it is liable to localize itself to the vicinity of the diseased vessel. In the case of secondary thrombosis, a pyæmic condition is often shown by repeated chills, and by high temperature. The rigors, according to Pitt, usually usher in the condition, in fact they may appear suddenly. The temperature is usually high, but is subject to marked depressions. The pulse is small and thready. Sometimes, there is great pain in the back of the neck. Eye symptoms are noted. These consist of strabismus, nystagmus, and optic neuritis. The latter is now conceded by experienced clinicians as of rather frequent occurrence, serving, indeed, as a valuable differential sign in the diagnosis of thrombosis and abscess. In the latter condition, neuritis is very exceptional, though it is otherwise stated by numerous writers. Delirium is generally a prominent symptom. Paralyses and contractures sometimes occur.

A valuable aid in the diagnosis of the disease is afforded by the œdema localized over the area of distribution of the veins emptying into the affected sinus. Unfortunately, this symptom is not as constant as we could wish. In thrombosis of the longitudinal sinus, the veins running from the temples to the anterior fontanelle are distended, and there are facial

cyanosis limited to the above-mentioned region and epistaxis. Thrombosis of the lateral sinus is sometimes attended by œdema back of the mastoid process, and nearly always by tenderness on pressure over the point of emergence of the mastoid veins, and extending down and behind the ramus of the jaw over the internal jugular vein. In thrombosis of the cavernous sinus, the ophthalmoscope reveals fulness of the retinal veins, and there are œdema of the eyelids and conjunctiva, and exophthalmos. In thrombosis of the internal jugular vein, there may be fulness and pain on the corresponding side of the neck.

The frequency of thrombosis of the lateral sinus calls for more extended remarks than have been already made. In general, it may be said that when a chronic otorrhœa suddenly disappears, and there come on severe otalgia, headache, rigors, and high temperature, thrombosis of the lateral sinus may be suspected. The œdema over the mastoid already mentioned, is not as valuable a sign as we could wish, for it is sometimes absent, and is not infrequent in inflammation of the mastoid cells, when the lateral sinus is unaffected. The trouble is, however, very liable to be accompanied by an inflammation of the veins leading from the sinus, and this causes local tenderness over the internal jugular vein in its upper third, and in the upper third of the posterior cervical triangle. This pain is found, on pressure, to be quite acute, and will often arouse the patient from his stupid state.

**Diagnosis.**—The diagnosis of sinus thrombosis must be uncertain in any event. It must always be suspected in any case of cerebral disease arising in conjunction with chronic suppurative ear disease. It is necessary, however, to differentiate it from cerebral abscess, meningitis, and mastoid inflammation, which subject will receive full consideration in the chapter on abscess.

**Prognosis.**—Thrombosis of the cerebral sinuses runs a course of from a few days to seven or eight weeks. The prognosis is always unfavorable, still it is believed that quite a number of cases of the primary form recover. Thrombosis of a single sinus is not necessarily fatal. If the constitutional condition which gives rise to it improves, recovery is by no means impossible. If, however, other sinuses participate in the trouble, the outlook is almost necessarily unfavorable.

Thrombosis of veins, by interfering with the nutrition of certain areas of the brain, leads to permanent changes in that important organ.

Thrombosis of infective origin runs a rapid course. It is curable by operation, but only when taken in hand before systemic infection has occurred.

**Treatment.**—Medicinal treatment directed to the thrombosis accomplishes nothing. In the case of marantic thrombosis attention to the general condition of the patient accomplished more than anything else. Stimulants and general hygiene are important. Surgical measures are



only applicable in the infective variety, and consist of thorough removal of every possible source of infection. The operation must be undertaken on exploratory principles, however. Should jugular thrombosis be established, that vein must be ligated in the neck, and the affected sinus opened and thoroughly cleaned according to antiseptic principles.

## THROMBOSIS OF THE CEREBRAL ARTERIES.

Thrombosis of the cerebral arteries is a condition in which the calibre of the vessel is obliterated by the formation of a clot at the seat of the obstruction.

**Etiology.**—Thrombosis is due to the disease of the walls of the arteries, generally to atheroma or syphilitic endarteritis. In the latter condition, there is a progressive narrowing of the calibre of the vessel by the specific inflammatory deposit; but this rarely increases to the extent of producing complete occlusion. Other causes are found in acute and chronic alcoholism, and in certain blood states, and cardiac asthenia.

**Pathology and Morbid Anatomy.**—Aside from the formation of the clot *in situ* and the disease of the vessels, the pathology of cerebral thrombosis differs but little from that of embolism. The subsequent changes in the affected areas of the brain are the same as in embolism.

**The Symptoms** closely resemble those of embolism and hæmorrhage; indeed, their differentiation is very often impossible. Prodromic symptoms are of frequent occurrence and consist of headache, vertigo, sensations of numbness and formication, hemiparesis, etc. In syphilitic cases, the headache is apt to be worse at night. These symptoms may exist but a few hours, or for several months.

The rapidity with which the attack is ushered in varies in different cases. It may reach its height within a very short time, or again it may take several days. Aside from the rapidity of its onset, its symptomatology is the same as in embolism and hæmorrhage.

**Prognosis.**—This is unfavorable, both as to life and recurrence should the patient be so fortunate as to rally from the first symptoms.

**Treatment.**—This must be conducted on the same principles as guide us in the treatment of cerebral hæmorrhage. Cardiac stimulants, though theoretically indicated by the physiological school, accomplish little or nothing.

## INTRACRANIAL TUMOR.

**The Etiology** of intracranial tumors, as that of new growths elsewhere, is but imperfectly understood. Attendant upon their existence, as of interest, are certain facts, the actual value of which from an etiological standpoint remains unknown. Sex seems to exert some influence as an etiological factor, for, with the exception of sarcomata, cerebral tumors occur with far greater frequency in males than in females, the proportion being about as two to one. Age likewise exercises some predisposition, for about three-fourths of the cases occur in patients of less than forty years of age, *i. e.*, children and adults in the prime of life. Age bears an important relation to the character of the tumor. Thus the vast majority of cases occurring during childhood are of tubercular nature. While tumors of this class may occur at any age, three-quarters of them are observed in patients of less than twenty, and one-half in patients of less than ten years. Gliomata likewise show a predisposition to affect young subjects, but here the greatest frequency is observed between the ages of twenty and forty.

Traumatism plays an important part in the production of many cases, although the relation between injuries and tumor is by no means as direct as that between the former and abscess. Many authors place but little reliance upon traumatism as a cause. The time intervening between the reception of the injury and the appearance of the tumor presents the widest possibilities in range. Usually the symptoms manifest themselves within a few months after the injury. In 1889 I saw, with Dr. C. R. Norton of this city, a case of sarcoma of the dura mater, in which the growth had resulted from a fracture of the internal plate of the skull some forty years before. The relation of cause and effect was thoroughly established, for the tumor was found attached to a spicule of bone projecting into the brain. Injuries undoubtedly act as an exciting cause of tumor in those constitutionally predisposed.

Of the constitutional causes at the foundation of cerebral tumors, three stand forth prominently. These are: (a) syphilis, (b) tuberculosis, (c) the dyscrasia produced by the formation of malignant growths in other portions of the body. Syphilitic tumors practically always appear as a result of acquired syphilis. They may occur at almost any period after infection. Tubercular tumors generally affect patients exhibiting a debilitated constitution, a tubercular inheritance, or those with tuberculosis elsewhere. Certain malignant growths in the brain may occur secondarily to the formation of similar tumors in other parts of the body.



Measles and other exanthemata occasionally serve to excite any dormant tendency to tumor formation.

**Pathology and Morbid Anatomy.**—The tumors observed within the cranial cavity are, in 90 per cent. of the cases, tubercular, syphilomata, gliomata, sarcomata, and carcinomata. Osteomata, fibromata, lipomata, and parasitic growths may also invade the brain and its membranes, but they are exceedingly rare in this location.

Intracranial tumors vary greatly in size in individual cases. Some varieties exhibit a tendency to greater growth than others. They also show a predilection for certain portions of the brain. Tumors which invade vital portions of the brain can never attain the size of which those attacking the so-called silent areas are capable. It is highly probable that the size of the growth depends more upon its opportunities for growth than upon the nature of the tumor *per se*.

The damage done to the brain by tumors is of a manifold character. Adjacent cerebral structure is damaged either by the infiltration of the normal brain structure by the tumor, or by the destruction of the same by pressure. Pressure in many cases is likewise exerted on distant structures, and thereby causes further damage, the gravity of which decreases with the distance from the lesion. In some cases the pressure is so exercised as to lead to the production of internal hydrocephalus by reason of closure of the interventricular foramina. Lastly tumors excite damage by irritation of surrounding structures, generally of the meninges, and this may amount to the actual inflammation.

Tubercular tumors present on section a cheesy appearance. They are regular in shape, and are generally surrounded by a softened layer of brain matter. They can therefore be readily lifted from their beds. They are always circumscribed, and very frequently multiple. While always serious, in some few instances they cease to grow. In such a case, the tumor becomes surrounded by a fibrous capsule, or, in other instances, it undergoes calcification.

Syphilitic tumors present some similarity to those just considered. They are, however, more irregular in shape, and the cheesy appearance on section is by no means so uniformly distributed throughout the mass. Their growing surface presents a grayish and gelatinous appearance. They do not infiltrate the surrounding structures. Under treatment they tend to disappear, becoming smaller and harder and of fibroid structure. They are almost always attached to the pia mater, a characteristic that should always enable one to differentiate them from tuberculomata.

Sarcomata may originate in any of the intracranial structures, bones, membranes, or brain. Starting in the cranium, they may soon perforate the skull and appear externally. They may attain to considerable size.

Gliomata constitute, according to some pathologists, but a variety of

sarcomata. These growths are peculiar to the central nervous system. They frequently infiltrate the brain substance. Their color and consistence are so frequently those of the brain itself, that it is sometimes almost impossible to detect their presence without resort to the microscope. They sometimes undergo central softening, and become transformed into cysts.

Gliomata are not infrequently invaded by extravasations of blood. They are liable to this accident because their substance is traversed by numerous thin-walled vessels.

**Symptomatology.**—The clinical pictures, presented by cerebral tumors, are remarkable for their variations. In some cases, the symptoms are noteworthy because of their severity, and in others, for their mildness or comparative absence. In exceptional instances tumors have been found post-mortem, when their existence was not even suspected during life. Notwithstanding these great discrepancies, the symptomatology of intracranial tumors is sufficiently characteristic to make their recognition a much easier task than in the case of many other organic diseases of the central nervous system. The symptoms, produced by cerebral tumors, may be divided into two classes, viz., the diffused, or those common to brain-tumors in general, and the focal, or those dependent upon the location of the growth.

Of the diffused symptoms, headache is the most constant; so constant, indeed, as to be practically never absent. Dana, however, disagrees with this statement, and says that headache is present in from one-half to two-thirds of the cases. The main characteristic of the headache associated with brain tumor is its constancy. It may be severe, indeed it generally is, or its progress may be marked by paroxysmal exacerbations, yet it is always present to some extent. Its location does not bear any definite relation to the site of the tumor in many instances. In others, the situations of the growth and the pain correspond very closely. The rule governing this point seems to be, that the nearer the tumor approaches the surface, the more likely is the pain to be localized and to indicate the situation of the growth. Localized tenderness is not apt to be present unless the meninges are affected by their proximity to the tumor.

Vomiting is frequently present, though by no means as constantly so as headache. It is especially frequent in cases of cerebellar tumor. It partakes of the well-known projectile character of this symptom when dependent upon brain disease.

Double optic neuritis constitutes a most important symptom of intracranial neoplasms. In fact, its occurrence in association with the headache and vomiting already described, I should consider as all but conclusive evidence of the presence of cerebral tumor. According to Gowers, it is found in fully 80 per cent. of the cases. It is especially constant in

tumors of the base of the brain; while, in tumors of the convexity, it is so frequently absent that the negative evidence thus afforded counts for but little. It occurs frequently without the slightest impairment of vision. It may appear early in the course of the disease, and disappear while the tumor continues its destructive career; or its advent may be delayed until the final stages have been reached.

Vertigo is frequently present, probably in about one-half of the cases.

The presence of a cerebral tumor is in perfect consonance with entire preservation of the mental faculties. Still, in many cases, especially when the tumor invades the frontal lobes, the mind gives evidence of weakening. The most characteristic of the mental defects are hebetude, somnolency, childishness, mental irritability, loss of memory, and power of attention. Speech may be disturbed, especially when the tumor is at the base of the brain, or in the vicinity of the speech-centres. In the final stages of the disease, stupor and coma frequently appear.

Other diffused symptoms of cerebral tumor are epileptic attacks which may consist either of general convulsions or of seizures of petit mal, general muscular weakness, and a variety of paræsthesiæ.

The focal symptoms of brain tumor may be divided into the (*a*) irritative, and (*b*) the destructive. Among the former, the most characteristic are the localized convulsive seizures, the so-called Jacksonian epilepsy. Examples of destructive symptoms are the paralyses dependent upon destruction of or pressure upon motor and sensory tracts. Thus we have produced hemiplegia, paralyses of cranial nerves, hemianæsthesia, aphasia, hemianopsia, etc., the full significance of which will be indicated in a subsequent portion of this work treating of the localization of the lesion.

The clinical course of a cerebral tumor is as a rule gradually progressive. Frequent exceptions are, however, observed. Very exceptionally the tumor exists for a long time, unaccompanied by disturbance of health, when without apparent reason, symptoms suddenly appear and death closes the scene. Very frequently the growth acts as a foreign body exciting irritation and even inflammation in neighboring structures, thus giving rise to quite a variety of symptoms which disappear as the inflammation or irritation subsides. The course of gliomata is apt to be interrupted by sudden apoplectic seizures resulting from the extravasations of blood into their substance. Some few tumors exhibit periods of latency and activity. This is especially true of tuberculomata and sarcomata.

**Diagnosis.**—The complete diagnosis of cerebral tumor involves the determination of three points, namely, the existence of the tumor, its nature, and its location. In the great majority of cases there is but little danger of confounding brain tumor with other conditions; the gradual progression of symptoms, the headache, vomiting, optic neuritis, convul-



sions or paralyses, should protect from all error. Especially do the above phenomena become significant when combined with focal symptoms; in fact, it is the combination of the diffused and focal symptoms that make the existence of cerebral tumor an assured fact. The remarkable fluctuations in the intensity of the symptoms in some cases leads to the diagnosis of functional trouble, an error that may be guarded against if the pathology of the lesion is borne in mind. Organic diseases of the brain not infrequently awaken into activity dormant hysterical tendencies, especially in women. Many diagnostic errors have thus been occasioned. If the investigator bears this in mind, and also that the discovery of phenomena unquestionably belonging to organic diseases more than outweighs a host of symptoms suggestive of functional or hysterical trouble, there should be but little danger of regarding a case of brain tumor as one of purely hysterical disease. The contrary error, that of diagnosing functional disease as organic, is less frequently made. Here the duration and course of the ailment affords an invaluable guide. Cerebral tumors have an average course of two years; functional disorders rarely present severe symptoms as early in their career as do the organic, and are also of much greater duration. Their remission of symptoms are likewise decidedly more complete.

*Uræmic convulsions* sometimes assume the Jacksonian type, and partake of all the characteristics of this phenomenon when due to brain tumor. The coexistence of uræmic headache and the neuritic form of retinitis albuminurica serves to make a diagnosis a difficult matter. The history of the case and unusual care in the ophthalmoscopic examination, and the examination of the urine (this latter including frequent determinations of the actual elimination of urea) will lead to a correct conclusion.

Lead poisoning sometimes presents headache, blindness, optic neuritis, and epileptiform seizures and so may present a close symptomatic resemblance to tumor of the brain. The history of the case decides.

It is not often that one must differentiate tumor and *abscess*. Causative indications here furnish an important guide, the presence of middle ear suppuration or the history of recent head injury pointing very strongly to the latter. The diffused symptoms are equally severe in both affections, though more acute in their course in abscess. Focal symptoms are strongly indicative of tumor. Abscesses present greater fluctuations in their symptoms.

The diagnosis of the nature of the tumor cannot always be made; still it should be attempted. Tubercular tumors almost always occur in childhood and early life. They are liable to association with tubercular disease in other portions of the body, systemic poor health and a tubercular family history. A syphilitic growth is to be suspected, especially in males and when a history of syphilis is obtainable, when the patient

is a young adult, and there is considerable meningeal irritation. Gliomata are characterized by sudden exacerbation of symptoms due to hæmorrhage within their structure; irritative phenomena are prominent; and the family history of tuberculosis and the pre-existence of syphilis, and the association of malignant growths (sarcoma and carcinoma) elsewhere can be negatived. Carcinoma is suggested by the presence of tumors of this character in other portions of the body. The patient is generally over fifty years of age.

The diagnosis of the situation of the tumor is based on the principles of localization, to the article on which the reader is referred.

**Prognosis.**—The prognosis of intracranial tumors, if those of syphilitic origin are excepted, is very grave indeed. Aside from those removed by surgical means, a fatal ending is to be expected within a period of from one to three or four years. Their average duration is two years. Tubercular tumors occasionally cease to progress. Syphilitic tumors are frequently cured by suitable treatment. It must be borne in mind, however, that these growths sometimes produce secondary changes in the brain structures, which changes cannot be removed by any treatment.

**Treatment.**—The only efficient treatment for intracranial tumors is removal by surgical means. The growths amenable to this procedure are limited in number, probably less than 10 per cent. of all cases. To be successful the operation must be undertaken while the tumor is yet small, and before secondary cerebral changes have been started. Circumscribed tumors offer decidedly better surgical opportunities than do the infiltrating varieties.

Much can be done by the proper exercise of palliative measures to mitigate the patient's sufferings in every case. Means of this character must be carefully individualized according to the case in hand. Empiricism in palliation is as blameworthy as empiricism in other departments of therapeutics. In conjunction with these must be enjoined attention to every measure capable of raising the health standard. Syphilitic growths are very amenable to large doses of iodide of potassium, administered according to the direction given in the section on syphilis of the nervous system. Sarcomata are apparently relievable in a measure by this means, for thrice now have I encountered cases of sarcoma of the dura mater in which the exhibition of iodide of potassium in large doses produced such remarkable alleviation of symptoms, that it was fast becoming a conclusion that the growths were after all syphilitic. Sudden death in one instance and gradual relapses in the others dispelled the delusion. The delay occasioned in one of the latter was unfortunate, for the case was one peculiarly favorable to operation, which was delayed too long owing to the apparent success of medicinal measures. I cannot urge the perseverance in the use of the iodide too

strongly in cases in which there is a possibility of syphilis. In one case, I succeeded in accomplishing considerable, but finally abandoned the drug as useless. Relapse occurred. The patient died six months later in the care of another physician. Autopsy discovered a large gummatous tumor.

As to remedies, those that give greatest promise of a sphere of usefulness are *belladonna*, *conium*, *hydrastis*, *sepia*, *calcareo carb.*, *graphites*, *baryta carb.*, and *arnica*.

*How long should one persist in the medicinal treatment of brain tumors?* In syphilitic tumors, iodide of potassium undoubtedly produces marked effects, even to the extent of curing in some instances. This last statement has been denied by Horsley, Gowers, and others, without sufficient warrant I think. The possibility that any given tumor may be of syphilitic origin must always be borne in mind, and the iodide of potassium treatment instituted, before recourse to operation is had. The duration of the period over which this treatment should be carried should not be too long. Horsley says it should not be persisted in over six weeks, and Starr makes it three months, unless the symptoms grow rapidly worse, showing the futility of internal medication. Care must be taken that improvement taking place during medication is a real improvement and not an apparent one. To be of value, moreover, the improvement should be a decided one.

*What cases of brain tumor are suitable for operation?* In answer to this question it may be said that all tumors presenting symptoms enabling them to be definitely localized in a position from which they can be removed with a reasonable degree of safety are suitable for radical operation. Estimates made from the post-mortem room show such to be about 10 per cent. of all cases of brain tumor. All irremovable brain tumors in which the diagnosis of tumor is reasonably certain, and in which the symptoms are such as to cause suffering, should be treated by palliative operation, namely, the removal of a large section of the skull to reduce intracranial tension. By this means pain may be greatly relieved, and the experience of Horsley and Keen shows that blindness from optic neuritis and its secondary atrophy may be prevented. In cases operated by the former the neuritis usually began to subside within three weeks of the time of operation.

As to the administration of analgesics my experience seems to indicate that such measures are unwise. I believe that the morphia in one case, and the coal-tar derivatives in another had much to do with the subsequent collapse from the operations. Still collapse is very frequent after the removal of brain tumors, sufficiently so to lead Horsley to propose and adopt in practice the division of the operation into two stages, performed on different occasions. First he removes the bone over the desired area. At a subsequent operation, the tumor is removed. He claims that by this means he has been enabled to avoid shock entirely.



*How early in the progress of a case should operation be performed?* An operation should be performed as early as possible ; that is as soon as the diagnosis of tumor has been made, and its failure to yield to medicine recognized. The removal of brain tumor is not as dangerous as generally believed. Thus of fifty-five cases in which the completed operation was performed, but sixteen died. The possibility of mistaken diagnosis must always be entertained, for eminent neurologists have thus erred.

## ABSCESS OF THE BRAIN.

Abscess of the brain is practically always a secondary affection. Quite a number of so-called idiopathic cases have been reported from time to time; but most of these occurred in years gone by when pathological study had not reached its present degree of accuracy, at which time the importance of infection was not recognized. The most authentic case of this character with which I am acquainted is that reported by Dr. A. R. Thomas in *The Transactions of the Homœopathic Medical Society of the State of Pennsylvania for 1885*. The conditions to which abscess of the brain is secondary are chiefly traumatism and suppurative disease of the middle ear and mastoid cells. Still, it may occur secondarily to the acute infectious fevers, notably influenza. Metastatic and tubercular cases have also been reported. The relationship between an injury and the resulting abscess is, at first sight, quite a varied one; but, analyzing the cases carefully, one finds the same condition present in all, namely, an infected wound. The injury may be of a very slight or of a very severe character. The abscess may appear two weeks after the traumatism, or, again, its manifestations may be delayed indefinitely. Damar Harrisson reports one case in which the existence of the abscess was not recognized until fourteen years after the accident. So far as the physical nature of the wound itself is concerned, von Bergmann insists upon but one feature which, he claims, must be present in all cases, and that is, a break in the continuity of the external integument. It is not necessary, he says, that the cranial bones be fractured or their fibrous coverings injured. Pitt, on the other hand, makes injury to the bones themselves the *sine qua non*, and thinks that slight injuries have but little influence. Sight must not be lost of the fact that Pitt bases his opinion on cases seen in Guy's Hospital, all of which were brought into the hospital suffering from severe injuries, and in which the abscess developed immediately after the traumatism. Even though the brain substance be severely contused, no abscess will develop, according to von Bergmann, if the integrity of the scalp be preserved. The time after the reception of the injury at which the abscess develops, while varying greatly in individual cases, never appears in less than two weeks after the accident; usually it manifests itself within three or four months. This is an important point, as it aids in the differentiation of encephalitis and meningitis on the one hand, and abscess on the other. The wound infection may be so unobtrusive after the accident that the wound heals without apparent complication. Macewen, indeed,

has observed several such cases. The injuries most liable to produce cerebral abscess are punctured wounds penetrating to or into the bone, and punctured fractures of the skull; compound fractures with large open wounds are not all liable to be thus complicated, as their serious nature is speedily recognized and efficient treatment is early instituted. Pathogenic affections of the face and scalp, *e. g.*, erysipelas, are far more liable to produce thrombosis or infective meningitis than abscess.

Ear disease is responsible for probably one half of the cases of cerebral abscess. Almost always it is the middle ear that is the origin of the trouble. The primary suppurative disease rarely gives rise to brain complications until it has existed for a long time, because in acute cases the mucous membrane of the middle ear is still intact, and the underlying bone uninjured. Still exceptional cases do occur, and in these it is more than probable that infection is carried to the brain by the lymphatics, evidence favoring this view being found in the presence of enlarged cervical and post-auricular glands. Cases have even occurred in which the membrana tympani was intact and the pus was retained within the middle ear. In some cases traumatism seems to combine with the aural suppuration in bringing on the symptoms of cerebral abscess. Thus the latter have been known to develop very shortly after a "box" on the ear, or even after the operation of trephining the mastoid. The importance of the two factors in the etiology of brain abscess, traumatism and middle ear disease, cannot be overestimated as data for diagnosis, for the symptomatology of the disease is such as give us but little light in their absence.

A very small percentage of cases of cerebral abscess occurs in conjunction with tuberculosis, pyæmic processes, the specific fevers and nasal disease. Concerning the former of these factors, it is especially when the lungs are the seat of disease that there is some danger of cerebral suppuration supervening. But recently I saw a case of this character with Dr. Geo. W. Titman, of Mt. Airy. The patient had previously been seen by Dr. Goodno, who regarded the pulmonary condition as one of probable acute pulmonary tuberculosis. Recovery ensued after a three-months' illness, after which symptoms indicative of a lesion in the right motor area appeared. An operation performed by Dr. W. B. Van Lennep evacuated a large abscess in this locality. Bronchiectasis is, however, believed to be the most prolific etiological factor, so far as the lungs are concerned. Chronic nasal disease with caries or necrosis of the ethmoid acts mechanically in the same way as does middle ear disease.

Age certainly seems to have some bearing on the production of cerebral abscess. Traumatic cases are more liable to occur in early life. Ear disease, pyæmia and distant suppuration rarely cause abscess under the age of twenty years.

**Pathology and Morbid Anatomy.**—In traumatic abscesses there



is no definite relation between the site of the injury and that of the abscess. Usually, the latter will be found adjacent to the part injured. It is often separated from the surface of the brain by intervening apparently healthy brain tissue, or it may be accompanied by a meningoencephalitis. Sometimes it is associated with a suppurative meningitis. Abscess from middle ear disease is found almost exclusively in one of two localities, viz., in the temporo-sphenoidal lobe when the middle ear is the primary seat of disease; and in the cerebellum when the mastoid cells are the primary focus from which extension takes place. The abscess is practically always on the side corresponding to the diseased ear. In pyæmic and traumatic cases, the abscess is generally found in the frontal lobes or centrum ovale.

When recent, the abscess has no limiting wall or capsule, but is surrounded by softened brain tissue, which mixes with the pus. In old cases, the abscess becomes encapsuled. In one of my cases, the enveloping membrane was of leathery consistency and was surrounded by softened brain tissue. Abscesses are nearly always single, the only exceptions to this statement being found in those cases in which the brain trouble is secondary to suppurative disease in the lungs and pyæmic processes generally. The character of the pus will vary according to the acuteness or chronicity of the lesion. In recent cases, it is reddish white, and is mixed with debris of brain tissue; in old cases, it is creamy or greenish yellow.

Cerebral abscesses range from one-third of an inch to three inches in diameter.

In abscesses secondary to middle ear suppuration, it is not necessary that there be any bone disease whatever. Under such circumstances infection is transmitted through one of the foramina or bony canals.

**Symptomatology.**—The symptoms of abscess of the brain may best be studied by considering them under two heads, the acute and the chronic cases. Acute cerebral abscesses are ushered in by marked symptoms. In the first place, there may appear the characteristics of suppuration, namely, rigors and high temperature. These are soon followed by symptoms pointing to the brain as the focus of disease, *i. e.*, by headache, delirium, projectile vomiting, optic neuritis, etc. As the disease progresses and the terminal stage is about to appear, we have added convulsions, paralyses and coma. The typical case, as thus outlined, is subject to many exceptions. Any one of these symptoms may be absent. Even the phenomena especially indicative of suppuration may fail to materialize, and instead of fever the patient presents a normal or sub-normal temperature.

Abscesses arising from chronic suppurative inflammations of the ear pursue a more chronic course than do those of traumatic origin. They often, however, manifest themselves rather suddenly. A previously pro-

fuse discharge becomes less in quantity, and severe pain develops. This is frequently most intense in the vicinity of the affected ear, and spreads rapidly over the entire temporal region. Vomiting is not an uncommon attendant. The advent of cerebral suppuration is sometimes ushered in by a chill, which varies greatly in degree, ranging from a slight sense of chilliness to the most pronounced rigor. Very often its character is misinterpreted, especially when it is so violent as to be mistaken for a convulsion. When the chill is repeated, there is probably systemic infection. The temperature during the initial stage is but slightly above normal. Later, it becomes normal, or falls even to below that point. Von Bergmann states that, in some cases at least, the temperature of the head on the affected side is greater than that of the corresponding point on the other side, and cites cases in point, in which the differences on the two sides amounted to  $1.8^{\circ}$  F. Some few teach that fever is often a marked symptom, though the best authorities now regard the high temperature as evidence of existing complications, as meningitis and thrombosis. As the abscess increases in size, and exerts pressure on surrounding tissues, and produces in them inflammatory changes, cerebration becomes affected. The patient is dull, is unable to concentrate his thoughts, and answers questions slowly. Pulse and respiration are slow; especially do we find the pulse slow in cases of cerebellar abscess.

The pressure symptoms of abscess are essentially the same as those present in cases of brain tumor. They are, however, characterized by their insidious onset. In fact, the more insidious the mode of onset, the more likely are the symptoms to be due to abscess as compared with meningitis, tumor or thrombosis. Prominent among the pressure symptoms of the late stages of abscess stands headache. This is not apt to be as severe as in the case of tumor. It is variable in its intensity. Its most important modality is its tendency to exacerbation with the rise in temperature, at which time the pulse-rate may fall. Pitt makes the peculiar observation that the headache accompanying abscesses from disease of the middle ear is apt to be more severe than in the cases of abscesses coming on from other causes. The explanation of this is to be found in the fact that abscesses from middle ear disease are generally larger than others, and the intracranial pressure is correspondingly great. As in the case of brain tumor, the pain *may* occupy almost any position in relation to the seat of the disease; but far oftener than in tumor, it possesses a value as a localizing symptom. Other pressure symptoms are drowsiness, unconsciousness, coma, Cheyne-Stokes respiration, and slow pulse. All of these may come on to a marked degree, and then disappear entirely, deluding the physician into the hope that recovery has taken place, and that the diagnosis of brain abscess was a mistake. Optic neuritis which is so frequently a symptom of brain tumor is rarely present in chronic abscess. Gowers and some other authors

express their belief in the frequency of this symptom, in the case of cerebral suppuration.

The symptoms arising from the special location of the abscess are of comparatively little value, except in those rare instances in which they point to disease of the motor areas. Abscesses in the frontal, temporo-sphenoidal, and occipital lobes, rarely give rise to area symptoms. The special condition supposed to accompany disease of the occipital lobe is hemianopsia, and yet in but one case of cerebral abscess reported in this location, that of Janeway, has this disturbance of vision been noted. The infrequency with which abscesses give rise to focal symptoms is to be accounted for by the fact that the suppurative process is of slow growth; the consistence of the abscess is such as to make an equable pressure in all directions; its substance being soft, the pressure exerted is not so injurious as it might otherwise be; and lastly the abscess is usually situated in the white substance of the brain, a portion less important than the gray matter from a physiological standpoint.

Were we to rely upon the localizing symptoms, we would fail very frequently in our diagnosis. Fortunately a study of the etiological factors at work in each case is of value in this connection. Toynbee laid down the rule that disease of the middle ear was liable to be followed by abscess in the temporo-sphenoidal lobe; while in cases in which the mastoid process is involved, the intracranial suppuration will be found to be in the cerebellum. This law is not infallible. It is simply a statement of the usual sequence of events. McBride and Miller make the further observation that when the auditory nerve is involved the suppuration is probably beneath the tentorium. When the auditory nerve is not affected, the abscess is probably in the temporo-sphenoidal lobe.

**Diagnosis.**—The greatest difficulty is encountered in differentiating cerebral abscess, leptomeningitis, and sinus thrombosis. Other things being equal, a high temperature favors meningitis, and repeated rigors, infective thrombosis. The problem must always be a complicated one, however, as these conditions may coexist; especially is abscess apt to be associated with meningitis. In both meningitis and thrombosis, the pulse is apt to be rapid and weak.

**Prognosis.**—Cerebral abscess untreated by surgical means must have an inevitably fatal issue. Acute cases end fatally, as a rule, within two or three weeks. Chronic cases very often take on a latent stage, during which the patient is apparently well. It sometimes happens that the abscess becomes encapsuled, and gives rise to little or no trouble for many years. Such a result is so exceptional that it should not enter into one's calculations in deciding as to the proper course of treatment. Surgical treatment in the hands of Macewen, of Glasgow, has been very successful. Others do not seem to have obtained as favorable results.



**Treatment.**—The treatment of cerebral abscess is the preventive. Every traumatism of the head, however slight it may be, must be treated on strictly antiseptic principles. Infected wounds so situated as to interfere with proper drainage, must be enlarged and made aseptic. Every case of middle ear suppuration must be treated with all possible speed. These dicta strictly followed will make brain abscess a thing of the past.

When, however, the abscess has developed, surgical procedures only can rescue the poor unfortunate. In all cases from ear disease I believe it to be good practice to open the mastoid before proceeding to trephine. Asepsis is thus more certainly assured, and besides the appearances of the parts will afford us valuable information. Then, too, if pus is found here its evacuation may clear up the case; if nothing is found, and the symptoms are still serious, we are encouraged to proceed further. The technique of the surgical treatment of these cases is so intricate that its thorough consideration in practice must be referred to the neurologist and the surgeon.

## CEREBRAL PARALYSES OF INFANTS AND CHILDREN.

Infants and children may develop cerebral paralyses as a result of any of the pathological changes which may befall adults. Owing to the delicate structure of the infantile brain, the results of these lesions are very serious in their effects, and produce clinical phenomena of a very different character from those observed in the case of adults. A separate study of the cerebral paralyses of children is, therefore, of the highest importance. As in adults, paralysis must be regarded as merely a symptom. It may result from cerebral, spinal or peripheral lesions. Spinal paralyses are almost invariably due to acute poliomyelitis; peripheral, to traumatism; and cerebral, to changes to be shortly mentioned.

While not as frequently observed as infantile spinal palsy, cerebral paralysis is by no means an uncommon condition. Peterson's studies show it to bear a relation to the spinal type as one to two. The loss of power may involve one lateral half of the body (hemiplegia), both halves of the body (diplegia), or both lower extremities (paralegia). Very exceptionally, one extremity only (monoplegia) is paralyzed.

Cerebral paralyses may develop from lesions produced in intra-uterine life, or during or after birth.

**Etiology.**—Cases arising during intra-uterine life are nearly always dependent upon injuries or serious illnesses of the mother while carrying the child. Prominent among the latter stand typhoid fever, uræmia, fright, syphilis and alcoholism.

Instances arising during birth are more frequently the result of slow, tedious labors than of the traumatism of the forceps. Such cases are practically of traumatic origin, and are due to meningeal hæmorrhage. In cases of head presentation the extravasation is nearly always at the base; in foot and breech presentations, at the convexity (McNutt). The condition is especially apt to occur in first-born children, and particularly when the mother is well advanced in years.

The primary cause of the acquired palsies is probably, in the majority of cases, alterations in the vascular structures, leading to ready rupture. Many times arterial degeneration is a direct sequel of infectious diseases, as scarlatina, measles, typhoid fever, variola, etc. Scarlatina becomes an indirect cause also, by reason of its subsequent nephritis. Congenital syphilis causes some cases, the pathological change in such probably being thrombosis. Other causes of acquired cerebral paralysis are found in fright, the status epilepticus, reflex eclampsia and traumatism.

**Pathology and Morbid Anatomy.**—The primary morbid changes present in the cerebral palsies of childhood are not yet fully understood. The difficulty in their pathological investigation arises from the fact that but few opportunities for autopsy are afforded when the initial lesions are fresh. When the patients die, it is generally when they are well along in years, and secondary degenerations and sclerotic lesions are the prominent changes. It is known, however, that as in adults, hæmorrhage, embolism and thrombosis are the prime pathological causes of many cases. Others start as a meningitis or meningo-encephalitis.

In the old cases, parencephalus, and sclerosis are very common. The cavities are generally found on the surface of the brain, and often extend well inward, even reaching to the ventricles. Atrophy and cysts involving the convolutions are frequently observed. Some cases are due to a simple arrest of development—an agenesis.

**Symptoms.**—These necessarily must present considerable variety in different cases, owing to the liability of the initial lesion to attack any portion of the brain. Portions having a motor function are nearly always affected, hence motor symptoms are characteristic. The onset of the congenital cases is, of course, not observed. Cognizance is sometimes taken of the child's condition at once; while in others, nothing is suspected until a few days after birth, when convulsions occur. Those produced during birth are characterized by asphyxia from which the child is resuscitated with difficulty.

In the acquired cases convulsions constitute a prominent initial symptom. Especially are they observed in association with loss of consciousness in cases with a cortical lesion. The distribution of the spasmodic movements will correspond with the seat of the lesion. It may be general or confined to but one extremity or to one half of the body.

Paralysis is the characteristic symptom. It may appear suddenly as in the cerebral palsies of adults with loss of consciousness, or it may succeed immediately on the convulsive seizure. The face is very frequently involved in the loss of power, but rapidly regains its normal condition, so that in old cases, the fact that it was ever involved, may be readily overlooked. The leg regains its functions more rapidly than does the arm. Exceptionally the paralysis may clear up so completely as to make its existence a matter of doubt. Many cases of so-called idiopathic epilepsy are in all probability examples of infantile cerebral paralysis with subsequent convulsive seizures. In one of my cases, the only remnant of the disease was a decided loss of power of the tongue, with a marked tendency to use the left hand.

Speech defects are comparatively common. This is observed in the cases of those taken in infancy by tardiness in acquiring that faculty, and by aphasia or paralysis of articulation in those advanced in years. Aphasia offers a better prognosis than in adults, and is far more frequently associated with left hemiplegia.



The later stages of infantile cerebral paralyses are very frequently associated with rigidities and contractures of the paralyzed parts. In the lower extremities, this leads to various forms of club foot, but especially to equino-varus, and in the upper, to contractures of the flexor muscles of the elbow, wrist and fingers. Sometimes the adductors of the thighs are thus affected, leading to the peculiarity of gait known as "cross-legged progression." This latter condition is especially common in cerebral diplegias and paraplegias.

The deep reflexes are exaggerated. Those of the arm, the elbow and wrist jerks are readily obtainable. Ankle clonus is marked. Exceptions to this statement are found in cases in which the contractures are inordinately great.

Abnormal movements are observed in the paralyzed parts. The most commonly observed of these are choreiform, athetoid, and associated movements. These are believed to be the direct result of the degenerations in the pyramidal tracts.

The paralyzed extremities fail to develop properly. They are shorter in length and smaller in circumference than their fellows, and their depraved nutrition is shown by their lowered temperature and other evidences of deficient circulation. The earlier the age at which the disease sets in, the more marked the trophic disturbances. It is therefore believed that, unlike infantile spinal paralysis, the atrophy is one of retarded growth.

Probably one-half of the cases are subsequently affected with epilepsy. The convulsions may be in every way similar to those of the idiopathic disease, or they may conform strictly to the Jacksonian type. Attacks of petit mal may be observed.

Mental impairment to some degree is nearly always observed. In many instances this may amount to but insubordination, waywardness, or deficiency of moral sense, while in extreme cases, it is represented by a most pitiful idiocy. Some cases simply give the impression to the observer that the child is one not possessed of ordinary mental powers. The mental enfeeblement in all cases seems to be in direct proportion to the extent of the pathological process.

The mental defects are very frequently associated with cranial deformities. In the majority of cases this consists of a flattening of the skull on the side of the lesion; in others it amounts to actual microcephalus, asymmetry of the skull, abnormal arching of the palate, etc.

**Prognosis.**—The convulsions and the initial symptoms are often followed by death, as is the case of similar states occurring in adults. When the patient survives these, the prognosis as regards recovery from individual symptoms is very poor indeed. The epilepsy and mental deterioration in particular are apt to prove disastrous to a life of usefulness. Comparatively few cases live beyond middle life.

**Treatment.**—Inasmuch as many cases of infantile cerebral paralysis are the result of the convulsions, preventive treatment applied to this symptom is always an important matter. This will be discussed in the chapter on “Infantile Eclampsia.” The early treatment of the paralysis should be conducted on the same principles as guide us in the management of ordinary apoplectic seizures in adults.

Late phenomena call for symptomatic treatment and management. Curative remedies exert but little or no influence over the epileptiform seizures. One is, therefore, obliged to resort to palliative measures. Here we must resort to the bromides in selected cases. At the same time it must be remembered that *very many* of the patients are intolerant of these drugs, and their administration must be conducted only by one possessed of a full knowledge of their physiological action. Bromism is readily induced. The complete suppression of the attacks is rarely possible, even when extravagantly large doses are prescribed. One must be satisfied, therefore, with reducing them to a minimum, having due regard to the general effect of the drug as well.

Some cases thrive best on a properly selected diet. These mentally enfeebled patients are often exorbitant eaters. Their morbid appetites must be properly controlled, and improper articles of food forbidden. A vegetable and milk diet is probably the best in the majority of instances. Care in this direction is often sufficient to ameliorate the epileptic state most markedly.

For the mental enfeeblement no course is more successful than education under the supervision of instructors experienced in the care of the feeble-minded. Large schools are objectionable, as it is but rarely that their clients receive any attention amounting to more than mere kindness to the body. A select school with a limited patronage should be chosen whenever the circumstances of the family will permit of such a course.

Surgical intervention is of value in two stages of the disease: Early, when it is evident that the paralysis is due to meningeal hæmorrhage, and the situation of the extravasation is definable with a fair degree of accuracy; and later, for the correction of deformities. For the latter, orthopædic apparatus is often sufficient; aggravated cases require tenotomy. Trephining and excision of portions of the cortex of the brain is rarely, if ever, a successful remedy, even in cases of a marked Jacksonian type.

Prolonged medication when the symptoms have become stationary is useless. Prior to that time, medicines must be administered solely on constitutional indications. Syphilitic cases, it is needless to say, require *mercury* and the *iodide of potassium*; hæmorrhagic cases, *arnica*, *rhus*, *causticum*. *Calcarea*, *sulphur*, *baryta carb.*, *aurum et natr. mur.* and *lycopodium* are often called for by constitutional states. The efficiency of these remedies can only be secured after months of administration.

## DEGENERATIONS OF THE BRAIN.

(A) CYSTIC DEGENERATION OF THE BRAIN. In this variety numerous small cavities are found scattered throughout the cerebral mass, mainly, however, in that area where the terminal branches of the cortical and central vascular systems of vessels approximate, and hence in an area in which local nutrition may be readily reduced to a point lower than compatible with health. These cavities probably represent enlarged perivascular spaces. Senile atrophy of the brain with its usual shrinkage in size of the organ is present, and affords some explanation for the existence of the cavities. The ventricles are generally enlarged. It is not known that cystic degeneration of the brain produces any characteristic symptoms.

(B) CHRONIC PROGRESSIVE SOFTENING OF THE BRAIN. While the majority of cases of softening of the brain constitute a secondary process, certain rare cases in which the change is a primary one, are encountered. The softening is of gradual onset, and is almost certainly progressive in course. It involves the white matter solely in the vast majority of cases. The changes may proceed to almost any extent, from limitation to one or more small foci to diffusion through the entire cerebrum. The etiology of this disease is unknown. It has been observed with equal frequency in both sexes; but it is decidedly more common in persons of advanced years, that it is in those between sixty and eighty years of age. It runs a course of from two months to two years. No characteristic symptoms are present. This can be readily understood. Different cases will present different clinical pictures according to the distribution of the lesions, their size and their rapidity of progress. Destructive symptoms predominate over the irritative. A diagnosis of chronic progressive softening can never be made with any certainty. It is rendered probable, however, by the absence of symptoms pointing to other conditions having an unequivocal symptomatology, and by the age of the patient.

(C) CEREBRAL SCLEROSSES. These may be divided into classes according to their supposed pathological origins, and again according to their anatomical distribution. According to the former, we have (a) degenerative sclerosis; (b) inflammatory sclerosis; and (c) developmental sclerosis. According to the latter, we have (a) diffuse sclerosis; (b) miliary sclerosis; (c) tuberous sclerosis; and (d) disseminated sclerosis.

(a) *Degenerative sclerosis.* This comprises the largest class of cases of sclerotic cerebral changes. In it may be included (1) the secondary



degeneration following lesions affecting the projection systems of fibres; (2) toxic degenerations as from lead poisoning, the so-called lead encephalopathy; and (3) the sclerosis dependent upon vascular disease.

(b) *Inflammatory sclerosis.* This variety includes cases in which irritative inflammation has been developed about tumors, abscesses, hæmorrhages, foreign bodies, etc., and after injuries. The latter cause is undoubtedly a very prolific one. The influence of injuries in the production of epilepsy, and other serious nervous diseases months after the accident finds its explanation in the production of a diffuse inflammatory sclerosis.

According to the anatomical distribution of the sclerosis we have as already stated:

(a) *Miliary sclerosis.* This is a condition first described by Drs. Batty Tuke and Rutherford. It consists of numerous minute spots of degeneration found only under the microscope. There has been considerable discussion relative to the genuineness of these lesions, some authorities, as Gowers and Savage, claiming that the morbid appearances are the direct result of the methods employed in mounting the specimens, and are in no sense evidence of pathological changes in the brain. Most cases have been described as found in insane patients. Gowers describes a case of miliary sclerosis in which minute nodules were found at the junction of the centrum ovale and the gray matter of the cortex, and at the outer side of the lenticular nucleus and the claustrum. In appearance these sclerotic foci resembled closely those of insular sclerosis. Osler describes a condition in which "on the surface of the convulsions there are small nodular projections, varying from a half to five or more millimetres in diameter. Single nodules of this sort are not uncommon; sometimes they are abundant. So far as is known no symptoms are produced by them."

(b) *Diffuse sclerosis.* Diffuse sclerosis may involve either the white or the gray substance of the brain, a part or a whole of one hemisphere; it may invade the cerebrum or the cerebellum. One variety of diffuse sclerosis of the brain is without doubt secondary to changes in the nerve elements themselves. In this there is diminution in bulk of the affected part, and the cortex is mainly affected. There seems to be a combined atrophy and sclerosis. The cortex is injured by hæmorrhages, inflammations, injury, or repeated epileptic seizures, failure of nutrition of the nerve-cells and subsequent atrophy follows, and then overgrowth of connective tissues takes place.

Another variety of diffuse sclerosis is probably syphilitic. In such cases (generally hereditary) the appearances to the naked eye are normal. The affected part feels indurated to the touch. The lesion involves principally the gray matter of one or more convolutions. The white matter nearly always escapes in hereditary syphilis.

Diffuse sclerosis of the brain has been observed in connection with chronic alcoholism, interstitial nephritis, etc.

Diffuse sclerosis of the brain can present no well-defined symptomatology. Occurring in children, the main clinical evidence of its existence is found in mental impairment, and even imbecility. The motor and sensory disturbances may include those appearing in any other cerebral affection.

(c) *Tuberous sclerosis*. In this variety, firm opaque white masses appear in and project beyond the convolutions.

## DISSEMINATED SCLEROSIS OF THE BRAIN AND SPINAL CORD.

**Synonyms.**—Insular sclerosis; multiple sclerosis; sclerosis *en plaques disséminées*.

**Definition.**—A chronic degeneration of the brain and spinal cord in which the pathological changes are scattered in foci of different sizes throughout the central nervous system; and characterized clinically by paralysis, tremor, ocular symptoms and other phenomena, varying according to the distribution of the lesions.

**Etiology.**—Heredity seems to play a rather more important part in the production of disseminated sclerosis than it does in other degenerative affections of the cerebro-spinal axis. The transmission is not a direct one, however; but consists in the inheritance of a neuropathic constitution, which acts as a powerful predisposing factor. The neurotic origin is further shown in those cases which occur, apparently, as the result of prolonged anxiety. Most cases occur in young adults, that is, in patients between the ages of twenty-five and forty years. Still, old age and childhood are not exempt. The disease has occasionally been observed to follow severe traumatisms, and that, too, when no serious cerebro-spinal symptoms followed immediately upon the accident. Syphilis has been assigned as a cause. Investigations show that it is seldom operative in this relation. Some cases occur from exposure to cold. The various infectious fevers, notably typhoid and typhus fevers, diphtheria, smallpox and erysipelas. It is believed that these act by exciting myelitic foci, which subsequently undergo sclerosis.

**Pathology and Morbid Anatomy.**—The characteristic lesion consists of numerous scattered foci of degeneration, ranging in size from one-twenty-fifth of an inch to one inch in diameter. Microscopically, they are irregular in shape, and are of a grayish-red color. Their outlines are sharply defined. In consistence, they are firmer, often decidedly so, than the surrounding normal nervous structure. They are found mostly scattered through the white matter in the brain. They may also invade the central ganglia, the crura, the pons and the medulla. When the spinal cord is involved, as is generally the case, any of its tracts or

systems may suffer. Microscopically, the lesions are found to consist of an overgrowth of neuroglia and a wasting of the proper nerve elements. The axis cylinders persist until an advanced period of the process of degeneration. This fact has afforded one explanation of the peculiar so-called tremor of disseminated sclerosis, it being alleged that motor impulses travel through the diseased area with difficulty, and hence give rise to irregularities in the motor impulses during efforts at voluntary movement.

**Symptomatology.**—When one bears in mind the remarkable variations possible in the distribution of the focal lesions, the equally remarkable variations in symptoms of the disease must be at once recognized. So great, indeed, are they, that one might be almost justified in declaring that disseminated sclerosis of the brain and cord has no characteristic symptomatology. Still, it has some remarkable symptoms, none of which can be said to be pathognomonic; but which, taken as a whole, make up a clinical entity not to be confounded with any other condition.

First among these symptoms comes paralysis. This appears early, not as an actual loss of power, but simply as a weakness of this or that particular portion of the body, especially, however, of the legs. Sometimes the paralysis is limited to one lateral half of the body; sometimes to the lower extremities; and sometimes it amounts to a simple monoplegia. With the lapse of time, it becomes better defined, and is finally recognizable as an absolute loss of power.

The next important symptom to note, and the one which above all others has had the most note made of it, is the so-called tremor. This consists of irregular movements of a part during efforts at voluntary motion. The amplitude of these movements is large, they are never rhythmical as in paralysis agitans. They cease absolutely while the patient is at rest. I feel that they are better characterized as jerking rather than tremulous in character.

Ataxia of movement is frequently observed. This is to be expected from the dissemination of the sclerotic foci through the different structural systems of the spinal cord. The many other symptoms associated with the ataxia, but especially the jerky "tremor," should prevent confusion with locomotor ataxia.

Eye symptoms are prominent. They consist mainly of amblyopia, nystagmus, and diplopia. Of these, the nystagmus is generally regarded as the most characteristic. It is probably present in two-thirds of the cases. The disorderly movements like the "tremor" are aggravated during movements of the eyes. The amblyopia presents varied characteristics according to the situation of the producing sclerotic lesion in the visual tract.

The disturbances in articulation are highly characteristic when



present. These consist of what has been denominated "scanning speech" or "syllabic utterance." There is apparently an ataxia or jerkiness of the muscles of articulation, and this leads to slow or studied utterance on the part of the patient to insure his being understood. Syllables and words are unduly separated in conversation.

Sensory symptoms are not wanting. The patient frequently experiences vertiginous sensations. Anæsthesia occurs whenever the sensory tracts are invaded by the degeneration. Sometimes the patient experiences dull pains in different portions of the body. Still sensory disturbances never become as prominent as the motor, for the reason that this function is apparently much more easily compensated for, and consequently requires a severer lesion for its destruction. Sometimes, though but rarely, a girdle sensation is experienced.

The patient may present almost any type of mental change. Usually the intellect is dulled and the memory weak. It has been said that the patients display good temper. This statement is to be doubted, for it has been my experience to find them irritable. The remarkable amelioration of symptoms in the course of the disease undoubtedly explains the patient's placidity in the midst of a very serious condition of affairs.

Certain irregular accompaniments of the paralysis are to be noted. Thus there may be muscular atrophy from involvement of the ganglion cells of the anterior cornua; in other cases there may be marked rigidity from implication of the pyramidal tracts.

The knee-jerks are usually exaggerated. Ankle clonus is often prominent.

**Course; Duration, etc.**—No organic disease of the nervous system presents such a remarkable course as does disseminated sclerosis. Its chronicity is proverbial, lasting as it does from five to fifteen years. More remarkable yet are the remissions of symptoms, remission that lead the uninitiated to the diagnosis of functional disease. Indeed, it is important to note that few cases of this disease go the rounds of the profession without receiving more than once a diagnosis of hysteria, and thereby an inferentially favorable prognosis.

**Diagnosis.**—It is usually taught that disseminated sclerosis is readily confounded with paralysis agitans. Reference to the clinical picture of the latter affection will show at a glance that the two diseases present hardly a point of resemblance. The great danger—and, moreover, a real one, for I have known eminent diagnosticians fall into the error—lies in the confusion of disseminated sclerosis with hysteria. The error is not to call hysteria an organic affection, but to diagnose disseminated sclerosis as hysteria. Due consideration given to the reflexes, the eye symptoms, and the jerky "tremor," should save the physician's reputation.

According to the situation of the sclerotic foci, disseminated sclerosis

must present similarities to locomotor ataxia, spastic spinal paralysis, dementia paralytica, bulbar paralysis, and meningitis. Differentiation is to be made by the presence of symptoms not possible of explanation with the existence of the respective lesions of these diseases.

Some cases in which all the classical symptoms of disseminated sclerosis were present have gone on to a fatal issue, and yet autopsies have failed to explain or find any abnormality. Such cases have never received satisfactory explanation.

**Prognosis.**—This is very unfavorable. So far as known, no case has been cured. The disease pursues a long, tedious course, ending fatally after a lapse of years.

**Treatment.**—No set treatment can be formulated, for cases differ so in their totality. One must be guided entirely by present indications. Electrical treatment is of considerable value when properly instituted, but it must be suited to the case, and the method must be altered from time to time as indications change.

As to remedies, the salts of *gold*, *lead* and *mercury* apparently have the greatest influence over the pathological process. Expecting nothing, one should be satisfied with little, and because a good remedy does not cure an incurable case, one should not flounder about among unknown and untried drugs trying to effect the impossible. I believe that more can be accomplished by the steady administration of a drug like one of the above, uninterrupted for months save by intercurrent remedies administered from temporary conditions, than by wild, unphilosophical empiricism.

## APHASIA.

A study of the subject of aphasia is beset with difficulties—difficulties made greater than they should be by the interest excited, and by the tendency of authors to manufacture plausible theories serving to hinder rather than to further the interests of truth. Standard dictionaries, terse though they be in their statements, are no less confusing in their information, for one finds on consulting them the widest variance in their authors' conceptions of the different varieties of aphasia. While dogmatism is contrary to all scientific principles, the writer feels that he will add to clearness of his description of the aphasias if he prefaces this section with definitions and dogmatic statements. Theories he will endeavor to avoid entirely, and confine himself to a consideration of clinical phenomena and, so far as known, their anatomical substrata. Let us now proceed first with our definitions.

*Aphasia* is the loss of power of speech, by reason of disease of the speech centres in the cerebrum. It is not to be confounded with aphonia, or loss of speech from laryngeal palsy; or with anarthria, loss of speech from paralysis of the muscles of articulation.

*Apraxia* is the loss of power of recognizing the nature and uses of objects seen, heard, touched, smelled or tasted.

*Alexia* is the loss of power of understanding written or printed language.

*Dyslexia* is a term used with such a variety of meanings as to make it a medium of confusion rather than of enlightenment. Gowers describes it as a paroxysmally recurring difficulty in reading. Berlin defines it as a morbid dread of reading. Both authorities regard it as a serious symptom, believing it to be evidence of degenerative cerebral changes. Billings makes both it and alexia synonymous with word-blindness.

*Word blindness* is synonymous with alexia.

*Word deafness* is the loss of power of understanding articulate speech.

*Aphemia* is aphasia dependent upon "impairment or loss of power of co-ordinating the muscles of phonation so as to articulate certain words, unaccompanied by paralysis, insanity, or loss of the power of comprehending, reading or writing the words." This is also called *ataxic aphasia*.

*Agraphia* is the loss of power of writing speech, the muscles of the hand and arm remaining unparalyzed.

*Paraphasia* is the speaking of one word when another is intended.



It includes also the dropping of syllables of a word, the mispronunciation of words, and the misplacement of words in a sentence.

*Amnesic aphasia* is the inability to use a word from failure to recall it.

*Amimia* is loss of gesture power.

*Asemasia*, a term rarely used in medical literature, signifies a condition in which aphemia, agraphia and amimia are all present. *Motor aphasia* is the term ordinarily used to convey the idea of asemasia.

Speech processes in the cortex of the brain are sensory and motor; receptive and emissive. The integrity of both is necessary to speech. According as one or the other of these is involved, we have sensory or motor aphasia. The different varieties of these have already been mentioned in the preceding definitions, and will be more fully described shortly.

The lesions in all varieties of aphasia will be found on the left side of the brain in right-handed persons, and in the right half of the brain in the left-handed. The right half of the brain is capable of education as a speech centre. Accordingly, when a lesion in the left half has produced aphasia, the right sooner or later compensates for the loss. A second lesion occurring in the speech centres on the right side will produce permanent loss of speech.

The seat of lesion in aphemia is in the posterior part of the third frontal convolution of the left side, and in the adjacent part of the ascending frontal convolution. The island of Reil is ordinarily included in this speech area, though with insufficient reason, for, so far as is known, no lesion limited to this region has produced speech defect. The fibres from the motor speech area pass to the internal capsule. Interruption of these likewise produces aphasia, but this symptom is then likely to be but temporary, as compensation takes place rapidly through the commissural fibres to the right hemisphere. Usually, injury or disease of the motor speech centre abolishes the power of writing, and this independently of any affection of the movements of the arm. It is believed that there are association fibres between the speech and arm centres, and any interruption of these abolishes the power of writing. Any cortical lesion of the motor speech mechanism must necessarily interfere with one terminus of the association fibres, so agraphia occurs; but a subcortical lesion of the tracts to the internal capsule can interfere with articulate speech without interfering with writing. The loss of ability to read (alexia), occurring in connection with lesions of the motor speech mechanism is not constant. Its existence is largely dependent upon the personal equation of the patient. The uneducated can read only by making appropriate movements of the lips in following the printed page. It may be necessary even for them to read aloud. In such cases motor aphasia *produces* alexia.

Amnesic aphasia has been attributed by Wernicke to a lesion of the

fibres between the sensory and motor centres by disease of the island of Reil.

Word deafness is dependent upon a lesion in the posterior half or third of the first temporal convolution of the left side.

Word blindness arises from lesion of the left angular gyrus.

In the examination of aphasic cases, the following suggestions by Starr are of inestimable value:

"To examine an aphasic thoroughly it is necessary to test—

"(1) The power to recognize objects seen, heard, tasted, or smelt, and their use.

"This will determine whether the condition of apraxia is present.

"(2) The power to recall the spoken name of objects seen, heard, handled, tasted or smelt.

"(3) The power to understand speech and musical tune.

"(4) The power to call to mind objects named.

"This will test the integrity of the auditory speech area and of the association tracts between other sensory areas and the temporal convolutions.

"(5) The power to understand printed or written words.

"(6) The power to read aloud and to understand what is read.

"(7) The power to recall objects whose names are seen.

"(8) The power to write spontaneously, and to write the names of objects seen, heard, etc.

"(9) The power to copy and to write at dictation.

"(10) The power to read understandingly what has been written.

"These tests will determine the condition of the visual word memories in the angular gyrus, and of the connections between this area and surrounding sensory and motor areas.

"(11) The power to speak voluntarily, and if it is lost, the character of these defects.

"(12) The power of repeating words after another.

"This will test the integrity of Broca's centre\* and its associations tracts."

**MOTOR APHASIA.** In the beginning of an attack of motor aphasia there is loss of articulate, written and gesture language. Later gesture language, and ability to utter certain words, mostly emotional or ejaculatory, return. Thus the patient is able to say "yes" or "no," or utter an oath, when a connected sentence is impossible. He understands everything said to him; and he can not read. The mind is perfectly clear, so the patient fully appreciates his condition. Certain expressions cannot be made as a part of a conversation, while they can be uttered under emotional influences, or as singing.

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\* The base of the left third frontal convolution and the adjacent portion of the ascending frontal.

The majority of cases of motor aphasia recover a considerable power of speech, mostly through education of the right hemisphere.

SENSORY APHASIA. *Word deafness.* The patient cannot understand heard words; though he hears sounds perfectly. Speech is deranged; the power of recalling words is lost. The power of uttering words automatically or under the influence of emotions is greater than in the preceding variety. The greatest difficulty is encountered in the use of nouns. Indeed the patient may be unable to utter any word of this part of speech. When the necessity for use of one arises he paraphrases it. Thus he speaks of a knife "to cut with." In word deafness the patient is not aware of his errors in speech.

Word blindness may arise from different causes. It may accompany motor aphasia in persons in whom the motor speech mechanism is habitually used in reading. It may occur as independent symptom apart from other varieties of aphasia. It is then due to lesion in the left angular gyrus.

Mind blindness and mind deafness necessarily include word blindness and word deafness. The patient is unable to appreciate the nature and uses of objects seen or heard.

Agraphia occurs in conjunction with motor aphasia (aphemia). Under such circumstances the patient cannot copy or write from dictation. In word blindness the patient cannot copy. In word deafness he cannot write to dictation. Isolated agraphia apart from other varieties of aphasia has been known to occur, but it is exceptionally rare.



## CEREBRAL LOCALIZATION.

The successful practice of cerebral localization is possible only through a thorough knowledge of the practical anatomy and physiology of the brain, together with an understanding of the underlying principles of pathology. Empirical matching of symptoms in the patient with typical cases outlined in text-books is well-nigh useless, for the different focal lesions distribute themselves through the cerebral substance in kaleidoscopic variety. To succeed, one must possess anatomical, physiological, and pathological knowledge, and to these he must add clinical experience. The functions of certain areas are so well understood as to make the recognition of disease limited thereto a not very difficult matter in the majority of instances. Numerous cases, however, will be observed in which lesions of other portions of the brain are impossible of correct diagnosis.

**Anatomical Considerations.**—For clinical study, the brain may be considered as consisting of surface or cortex, the central white substance or centrum ovale composed of numerous systems of conducting tracts, and the basal ganglia.

The cortex is to be studied as consisting of lobes, lobules, convolutions, fissures, and sulci. These we will study *seriatim*.

Taking first the *lobes*, we find these to be four in number, the frontal, parietal, occipital, and the temporo-sphenoidal.

The *fissures* are the longitudinal, the Rolandic, the Sylvian, the parieto-occipital, the callosal-marginal, the calcarine, and the parallel.

The *Longitudinal Fissure* divides the two halves of the cerebrum.

The *Fissure of Rolando* starts from a point just posterior to the middle of the longitudinal fissure, and extends downwards for a distance of three and three-eighths inches at an angle of sixty-seven degrees. It reaches nearly to, but does not join the fissure of Sylvius.

The main portion of the *Fissure of Sylvius* runs in a nearly horizontal direction, and separates the temporo-sphenoidal lobe below from the frontal and parietal lobes above. Near its anterior extremity it gives off a small branch, described as the vertical or short limb of the fissure. Between the vertical and horizontal fissures is a portion of cortex known as the operculum.

The *Parieto-occipital Fissure* is seen mainly on the median surface of the brain, only its outer extremity encroaching on its convexity at a point midway between the upper terminus of the fissure of Rolando and the posterior extremity of the occipital lobe.

The *Calcarine Fissure* can be seen only on the median surface. It joins the parieto-occipital fissure at its innermost terminus, and extends backwards to the posterior extremity of the brain. The calcarine and parieto-occipital fissures and the outer edge of the median aspect of the brain bound a triangular area called the *cuneus*.

The *Calloso-marginal Fissure* is found on the anterior portion of the median surface of the brain. It lies midway between the upper cerebral margin and the corpus callosum. Its posterior extremity curves upwards.

The *Frontal Lobes* constitute that portion of the cerebrum lying

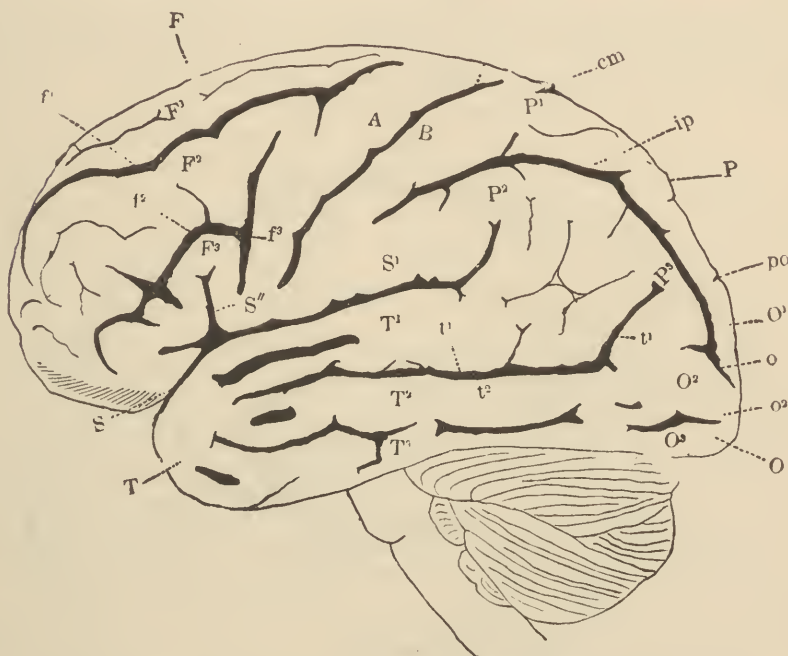


FIG. 30.—OUTER SURFACE OF THE LEFT HEMISPHERE. (After Ecker.)

F, Frontal lobe; P, parietal lobe; O, occipital lobe; T, temporo-sphenoidal lobe; S, fissure of Sylvius; S', horizontal, S'', ascending ramus of the same; c, central sulcus or fissure of Rolando; A, anterior central or ascending frontal convolution; B, posterior central, or ascending parietal convolution; F<sup>1</sup>, superior, F<sup>2</sup>, middle, and F<sup>3</sup>, inferior frontal convolutions; f<sup>1</sup>, superior, f<sup>2</sup>, inferior frontal sulcus; f<sup>3</sup>, precentral sulcus; P<sup>1</sup>, superior parietal or postero-parietal lobule; P<sup>2</sup>, P<sup>3</sup>, inferior parietal lobule, viz.: P<sup>2</sup>, supra marginal gyrus; P<sup>3</sup>, angular gyrus; ip, intraparietal sulcus; cm, termination of the calloso-marginal fissure; O<sup>1</sup>, first, O<sup>2</sup>, second, O<sup>3</sup>, third occipital convolutions; po, parieto-occipital fissure; o<sup>1</sup>, transverse occipital sulcus; o<sup>2</sup>, inferior longitudinal occipital sulcus; T<sup>1</sup>, first, T<sup>2</sup>, second, T<sup>3</sup>, third temporo-sphenoidal convolutions; t<sup>1</sup>, first, t<sup>2</sup>, second temporo-sphenoidal sulci.

anterior to the fissure of Rolando. The *parietal lobe* includes that portion of the brain lying above the Sylvian fissure and between the occipito-parietal and Rolandic fissures. The *occipital lobe* includes all lying posterior to the fissure last named. The *temporo-sphenoidal lobe* lies below the fissure of Sylvius.

Each of these lobes has its convolutions, named according to their position or to some peculiarity in shape. Thus in the frontal lobe we

find the *first, second and third frontal convolutions*, running in general direction parallel to the longitudinal fissure, the first in number being the nearest to that landmark. On the under surface of this lobe, that is the portion resting upon the orbital plates, we find adjoining the longitudinal fissure, the *gyrus rectus*, next to a convolution in which the olfactory lobe is set.

On either side of the fissure of Rolando are important convolutions, known generally as the *central convolutions*, the so-called motor area. The anterior one is called the *ascending frontal* or the *pre-Rolandic convolution*, and the posterior, the *ascending parietal* or the *post-Rolandic convolution*. Separating the ascending frontal convolutions from the ones in front of it is the *precentral sulcus*. The remaining important convolutions of the parietal lobes are the *supra-marginal* and the *angular gyri*. The supra-

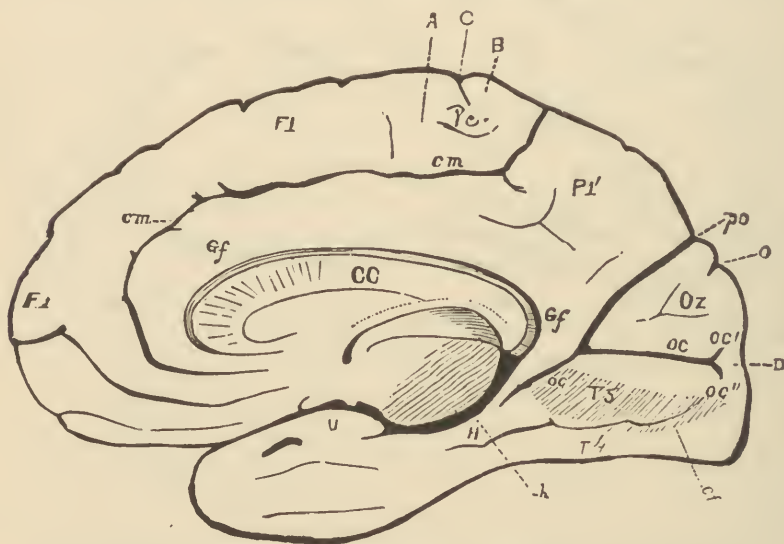


FIG. 31.—INNER SURFACE OF THE RIGHT HEMISPHERE. (After Ecker.)

CC, corpus callosum, longitudinally divided; Gf, gyrus fornicatus; H, gyrus hippocampi; h, sulcus hippocampi or dentate fissure; U, uncinate gyrus; cm, calloso-marginal sulcus; F1, median aspect of the first frontal convolution; c, terminal portion of the central sulcus, or fissure of Rolando; A, ascending frontal, B, ascending parietal convolution; Pe, paracentral lobule; P1', precuneus; Oz, cuneus; po, parieto-occipital sulcus; o, transverse occipital sulcus; oc, calcarine fissure; oc', superior; oc'', inferior ramus of the same; D, gyrus descendens; T4, gyrus occipito-temporalis lateralis (lobulus fusiformis); T5, gyrus occipito-temporalis medialis (lobulus lingualis); cf, collateral or occipito-temporal fissure.

marginal convolution lies just above the posterior extremity of the fissure of Sylvius. The angular gyrus is situated behind the posterior end of the Sylvian fissure, and extends backwards to the occipital lobe and upwards to the intra-parietal sulcus. Its ends join the first and second temporal convolutions.

The temporo-sphenoidal lobe has two, sometimes three convolutions. These run parallel to the Sylvian fissure. The sulcus dividing the *first and second temporal convolutions* is called the *parallel fissure*.



On the median aspect of the cerebrum we have two important convolutions in addition to those already mentioned. Between the callosomarginal fissure and the upper margin of the median surface is the *marginal convolution*. Between the same fissure and the corpus callosum is the *gyrus fornicatus* or the *convolution of the corpus callosum*. Continuous with the gyrus fornicatus and extending on to the temporo-sphenoidal lobe is the *hippocampal convolution*.

The *uncinate* and *lingual gyri* are on the median surface of the temporo-sphenoidal lobe. The uncinata is the upper one, and has been so named because its anterior extremity curves around like a hook. The lingual gyrus lies just below the calcarine fissure.

The *lobules* of the cerebrum are the paracentral, the quadrate, the cuneus, and the superior and inferior parietal. The quadrate is also called the precuneus; it lies between the occipito-parietal fissure and the upcurved extremity of the callosomarginal fissure.

The *superior parietal lobule* forms that portion of the parietal lobe into which extends the ascending parietal convolution, and lying above the interparietal fissure or sulcus. The *inferior parietal lobule* lies immediately below that fissure.

About the anterior end of the fissure of Sylvius is an area of unusual physiological importance, owing to its relations to the function of speech. The operculum has already been mentioned. Lying within the fissure of Sylvius and hidden by the operculum is the *island of Reil*, which consists of from five to seven small convolutions.

The *centrum ovale*, the white substance of the brain, consists of conducting tracts. These may be divided into three classes or systems: (1) projection fibres; (2) commissural fibres, and (3) association fibres. The projection fibres or tracts transmit motor and sensory impulses from the cortex to the spinal cord. The commissural fibres connect corresponding points in each lateral half of the cerebrum with each other. The association tracts run between adjacent portions of the cortex.

The *basal ganglia* are the optic thalami, the corpora striata, the corpora quadrigemina, the corpora geniculata.

Of these the corpora striata are of the greatest physiological importance. Each of these bodies consists of two masses of gray matter separated by a band of white. The former are known as the caudate and lenticular nuclei, and the band of white between as the internal capsule. This internal capsule is a portion of the projection system of fibres, for it contains the fibres extending from the cortex to the crura. It is divided into two portions, which are united by a sharp bend, the knee of the capsule. The anterior portion of the capsule transmits fibres from the frontal lobes only. The posterior limb contains the fibres from the motor area and the occipital lobe.

The *corpora quadrigemina* are masses of gray matter lying immedi-

ately above the aqueduct of Sylvius, and beneath the posterior portion of the corpus callosum. The anterior pair are the larger, and are called the nates; the posterior, the testes. They are connected with the optic thalami and the optic tracts.

The *corpora geniculata* are two masses placed "on the outer side of the corpora quadrigemina and on the under and back part of each optic thalamus, and named from their position, corpus geniculatum externum and internum. They are placed one on the outer, and the other on the inner side of each optic tract."

**Physiological Considerations.**—The functions of all the parts above mentioned has not, by any means, been sufficiently elucidated. Of quite a number of them, it may be said that we know absolutely nothing. There are some situations in which quite large lesions give rise to no symptoms whatever. These are generally referred to as the silent areas of the brain.

For the present, the frontal lobes must be regarded as belonging to these silent areas, for while we are well aware of the fact that they, in some way, subserve mental processes, we also know that they may be very extensively diseased without giving rise to any trouble.

The most interesting, probably because the best understood, is the motor area of the cortex. This as already stated is composed of the ascending frontal and ascending parietal convolutions, together with the upper extremity of the first frontal convolution, and the paracentral and superior parietal lobules. The muscles of the legs find their centres in the upper portion of this area, the arm centres being in the middle on either side of the Rolandic fissure, while the face finds central representation in the lowest of all. The expression above used, specifying a representation of the muscles of a part, does not exactly specify the correct idea, as it is not muscles but movements that are represented in the cortex.

In assigning to each convolution its appropriate functions a caution is necessary. While the surface of the brain is spoken of as consisting of fissures and convolutions, it must be remembered that these are but relative terms, and that the bottoms of the fissures and sulci will be found to contain the same layers of gray matter as surmount the convolutions. It must also be borne in mind that the sulci and fissures do not act as boundary lines for definite functional parts.

Taking, first, the leg centres, we find them occupying the convolutions bordering on the longitudinal fissure, both on the median and outer surface of the brain. Different authorities assign the centres for each movement somewhat differently. The centres for the hip seem to lie most anteriorly, that is, in the ascending frontal and upper portion of the first frontal convolution. Those for the knee and hip ankle occupy the upper extremity of the ascending frontal; and those for the great toe the

upper portion of the ascending parietal; and those for the smaller toes the space between the last-mentioned area and the parieto-occipital fissure.

The arm centres lie on either side of the fissure of Rolando, in the motor convolutions. Taking them from above downwards, they occupy the middle two-fourths of these in the following order: shoulder, elbow, fingers, index finger, and thumb.

The face centres are found in the very lowest portion of the motor convolutions, occupying mainly, however, the ascending frontal.

The centres for the trunk muscles, if we are to rely upon the teachings of Horsley and Schaefer, are to be placed upon the inner surface of the hemispheres. This statement is not to be accepted as positively reliable. Much uncertainty must exist as to these centres, for the reason that the functions subserved by these muscles are such as to require bilateral action, and, therefore, bilateral representation in the cortex. Of this, more will be said hereafter.

A centre for the upper eyelid is believed to exist in the angular gyrus.

The centre for the pharynx and the larynx has been assigned to the lowermost portion of the ascending convolution bordering on the fissure of Sylvius.

The situation of the sensory centres has not yet been positively determined. The weight of clinical and experimental evidence favors the view that the motor area is likewise a sensory one.

The most important function subserved by the occipital lobe is that of vision. That portion of this lobe, already described as the cuneus, contains the sight centres for the same-named half of each retina. Thus a lesion of the right cuneus causes blindness of the right half of each retina. Anterior to the occipital lobe, and in the angular gyrus, is a higher visual centre, in which both half fields find representation. Destruction of this area causes obscuration of vision in the opposite eye, with concentric limitation of the visual field. The eye of the same side seems to have representation also, for the lesion seems to damage the vision of that. The primary visual centres are located in the posterior part of the optic thalamus, the external geniculate body, and the anterior corpora quadrigemina.

The sense of smell finds its cortical centre at the anterior extremity of the uncinate convolution, on the same side as that on which function is abolished. The primary olfactory centre is the olfactory lobe.

The sense of taste has its cortical centre in the lingual gyrus.

The auditory centre is in the hinder part of the first and possibly of the second temporal convolutions. The primary centres are believed to be located in the posterior corpora quadrigemina and the internal geniculate bodies.



A consideration of the speech centres and the different varieties of aphasia has been presented in the preceding article.

**Principles Involved in Localization.**—As already stated in these pages, the correct understanding of a given case of cerebral disease involves the determination of two important points: the first is the nature of the lesion, and the second its location. A satisfactory conclusion respecting these can only be reached by a careful study and a logical consideration of all the data obtainable. The determination of the former is often of great importance in assisting to that of the latter point.

To illustrate: In the chapter on cerebral hæmorrhage it was taught that the most frequent cause of that trouble, indeed the usual cause, in people who have passed middle life, is rupture of miliary aneurisms, and this rupture takes place almost invariably in the lenticulo-striate artery. If, then, we have the conditions present showing such a pathological condition, and if we have complete hemiplegia, we are justified in diagnosing capsular lesion, although similar symptoms may arise from lesion elsewhere. Still another illustration may be found in connection with disease of the ear. If the symptoms point strongly to the presence of cerebral abscess, it is a matter of experience that that abscess is nearly always either in the temporo-sphenoidal lobe or in the cerebellum. If the mastoid is diseased, the cerebellum is more likely to be the seat of trouble.

All symptoms from focal brain disease are not of equal value for localization purposes. To aid in the consideration of the subject, symptoms may be considered as direct and indirect. The direct symptoms are those resulting from the actual damage done by the lesion within its limited area. The indirect symptoms are those arising by reason of the interference of the functions of distant or neighboring parts. This it may do by the very sudden onset of the lesion, the interference with the cerebral circulation usually occurring under such circumstances, generally exerting a profound and widespread impression; a tumor by its mechanical action compresses or irritates adjacent parts. In the case of hæmorrhage, the brain recovers from the shock, and the indirect symptoms disappear, leaving a symptom or series of symptoms dependent solely upon the local destruction by the clot. These only are of value for localization purposes.

It is important always to study the march of the symptoms. Taking a tumor in the motor area by way of illustration, let us presume that the convulsion first involves the thumb only. Later the spasms are more widespread. In subsequent attacks the movement covers increasingly larger areas because of the natural increase of the growth. Then come headache and general symptoms. The important symptom is the spasm of the thumb. It shows the starting point of the lesion.

The initial localizing symptom has been very appropriately called the signal symptom.

Lesions must be regarded as producing two classes of direct symptoms, the destructive and the irritative. In the clinical investigation of each case, it is necessary to assign every symptom to either of these classes.

It is important to bear in mind that muscles accustomed to act bilaterally have bilateral representation in the cerebral cortex. The muscles of the trunk, as are also those of the larynx and pharynx, are of this order. In case of a destructive lesion affecting the centres for these parts, but little if any noticeable loss of power results. The reason for this is found in the fact that the healthy hemisphere at once assumes the function of the injured one. Compensation takes place at once. Certain muscles have but partial bilateral representation; for example, those of the hip and shoulder joints. The legs have better bilateral representation than the arms; a condition one is led to expect, because the legs are accustomed to act in association as in walking. Highly specialized movements as those of the fingers have representation on one side only. When they are paralyzed, compensation is not likely to take place.

In convulsions dependent upon lesion of one hemisphere, muscles having bilateral representation are likely to be bilaterally affected.

**Lesions of the Frontal Lobes.**—It has already been stated that the probable function of the frontal lobes is mental. In unilateral lesions, but little psychical disturbance follows, as a rule, probably because the healthy side compensates for the loss on the other. When actual mental impairment is not prominent, there may be noticed some changes in the patient's character, as peevishness, inability to concentrate thought, inattention, etc. A lesion at the posterior part of the upper frontal convolution causes symptoms referred to the leg of the opposite side of the body. A centre for the movement of the head and eyes has been described as situated at the base of the second frontal convolution. A lesion here rarely produces permanent impairment of these movements. For the opposite side of the brain compensates rapidly for the loss. Some unsteadiness in walking similar to that observed in cerebellar trouble, has been observed as occurring sometimes in frontal lobe disease.

**Lesions of the Motor Area.**—According as the lesions are destructive or irritative, the symptoms are paralytic or convulsive. The distribution of these will vary according to the limitation of the lesion. Lesions producing localized convulsions are nearly always cortical. The cortical origin of such lesions becomes a certainty when the paroxysms are observed to recur from time to time, always presenting the same type. A convulsion from cortical lesion may spread and involve all the parts tributary to that side. In paralyzing lesions, on the other hand, the loss of power is in strict accordance with the destroying lesion.

Lesions of the visual area have been already described when treating of hemianopsia.

**Lesions of the Temporo-Sphenoidal Lobe.**—The only function at present assigned to these lobes is auditory. The posterior half or third of the first and possibly of the second temporal convolutions contains centres of hearing for the opposite ear. Destruction of this area produces deafness of the opposite ear. Irritation excites auditory hallucinations. Convulsions preceded by auditory aura have been caused by lesions in this situation. The deafness from unilateral destructive lesions is but temporary, as the healthy side rapidly compensates for the lesion.

Conjugate deviation of the head and eyes may occur in association with temporo-sphenoidal lesion, but such association is probably dependent upon auditory impressions.

A lesion in the uncinate convolution produces loss of smell on the corresponding side. If the lesion be irritative, olfactory hallucinations take place.

**Lesions of the Centrum Ovale.**—These must be diagnosed by exclusion, and in part by reasoning from analogy, as suggested by Starr. The symptoms are such as to exclude disease of cortex, pons or basal ganglia. At the same time they show involvement of association, projection or commissural tracts. On the other hand, it must be remembered that the centrum ovale is often the seat of extensive disease without any evidence thereof being present during life.

Convulsions rarely occur in central disease unless the lesion be immediately beneath the cortex. Aphasia may occur from central disease, but it is generally of short duration.

**Lesions of the Corpus Callosum.**—These produce no characteristic symptoms. In most cases of tumor in this situation, mental deterioration has been a prominent feature, but inasmuch as local softening in this situation has given rise to no symptoms, it is fair to presume than the symptoms occurring in connection with cases of brain tumor, depend upon the pressure exerted on other structures.

**Lesions of the Corpus Striatum.**—This ganglion may be considered as consisting of the caudate and lenticular nuclei and the internal capsule. Disease of the gray matter, if of such a character as not to damage the internal capsule, rarely produces symptoms. The only exception to this statement is found in the occasional association of mobile spasm with lesions in the posterior portion of the lenticular nucleus.

The internal capsule is, however, a very important structure, as it contains all the fibres from the cortex on their way to the crura. The functions of the anterior limb are not well understood as yet. Lesions in this situation are very rare. Lesion of the posterior limb produces hemiplegia, the lower face, the arm, and the leg on the opposite side



being paralyzed. Defect of speech occurs at first, but soon disappears. If the lesion is left-sided, there may be temporary aphasia. Convulsions are possible, though unusual phenomena. The paralysis is persistent, and is generally followed by rigidity of the affected limbs. The hemiplegia is not necessarily complete, as the lesion may involve only a few of the motor fibres, those going to the arm for example.

A lesion of the posterior third of the posterior limb of the capsule produces hemianæsthesia, and in some cases hemianopsia also as already stated. A partial lesion affecting the sensory fibres may excite widespread pains instead of anæsthesia.

**Lesions of the Corpora Quadrigemina.**—The anterior tubercles of the corpora quadrigemina have to do with the reflex movements of the pupil and ciliary muscle. The posterior tubercles and the internal geniculate body have some connection with the auditory nerve, and have apparently some fibres connected with the cerebellum. The nuclei of the third nerves lie immediately beneath these bodies. Lesions in this situation are therefore apt to cause ataxia of movement, pupillary inaction, and by extension of the lesion, motor oculi palsy. Blindness is sometimes present, but is due to optic neuritis.

**Lesions of the Crura Cerebri.**—The characteristic symptom of lesion of the crus is alternate hemiplegia of the motor oculi type. The eye muscles on the side of the lesion, and the face, arm and leg on the other, are paralyzed. Loss of sensation is sometimes present, as are vaso-motor disturbances.

**Lesions of the Pons.**—Several types of paralysis may ensue from unilateral disease of the pons. In one there is hemiplegia with paralysis of the fifth, facial or abducens. In another, when the lesion is high up in the pons, there is hemiplegia of the same distribution as observed in capsular lesion. In this case, there is loss of the power of conjugate deviation of the head and eyes to the side of the lesion.

**Lesions of the Cerebellum.**—Disease or injury of either lateral lobe of the cerebellum produces no symptoms. Lesion of the middle lobe produces inco-ordination of gait, simulating that of intoxication. The knee-jerks may be either absent or present. Sometimes they are found absent at one stage of the disease, and either present or exaggerated at another. Tumor of the cerebellum, and this is the most frequently encountered lesion in this locality, is apt to be associated with optic neuritis and vomiting and deafness. In many cases of optic neuritis, vision is either not at all or but slightly impaired. But in cerebellar disease, loss of vision seems to be more frequently observed. This probably finds an explanation by the pressure exerted on the anterior corpora quadrigemina. The deafness, when present, arises from pressure on the posterior corpora quadrigemina.

## CRANIO-CEREBRAL TOPOGRAPHY.

The application of our knowledge of cerebral localization in practice, requires that we have some means by which we can reach surgically the desired situation in the brain. Considerable time has, therefore, been spent in the study of certain landmarks on the surface of the skull and their topographical relation to the principal convolutions and fissures of the brain. The result of these investigations has been to give us means by which these different structures can be reached with a fair degree of accuracy.

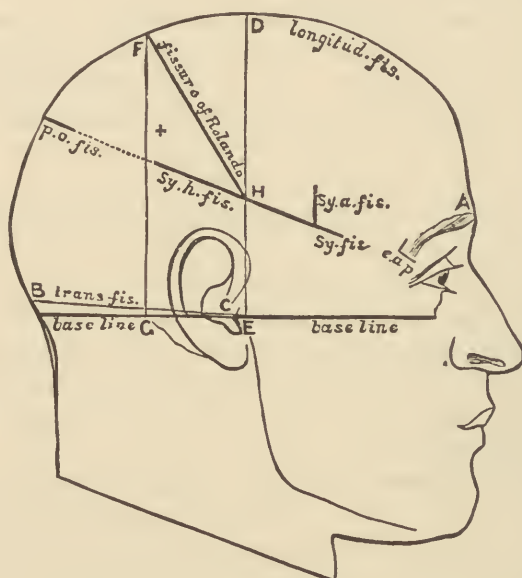


FIG. 32. (Reid.)

A, Glabella; B, external occipital protuberance; *e.a.p.*, external angular process of frontal; B C, transverse fissure; A B, longitudinal fissure; *Sy. fis.*, Sylvian fissure; *Sy. h. fis.*, horizontal limb of fissure of Sylvius; *Sy. a. fis.*, ascending limb of fissure of Sylvius; D E, perpendicular line from depression in front of external auditory meatus to middle line of top of head; F G, perpendicular line from posterior end of base of mastoid process to middle line of top of head; F H, fissure of Rolando; *p. o. fis.*, parieto-occipital fissure; +, most prominent part of parietal eminence.

The most important point, from a surgical view—important, because of the well-established knowledge of the adjoining convolutions—is the fissure of Rolando. This may be located on the scalp by several methods. The most convenient is that of Thane. He takes a point on the median line, one-half inch posterior to the middle of a straight line from the glabella to the inion. This marks the upper extremity of the fissure of Rolando. To determine the further situation of the fissure, a line is drawn from this point downwards and forwards, at an angle of sixty-seven degrees, the upper three and three-quarters inches of which mark the fissure.

Wilson has devised a special instrument, called the cyrtometer, for

determining the position of the Rolandic fissure, but my experience with it makes me prefer the tape-measure, and a graduated arc such as comes with school sets of geometrical instruments.

For determining certain other points on the surface of the brain, Reid's method is the best (see Fig. 32). The longitudinal fissure is first located by drawing a line from the glabella to the inion. The next point to determine is the situation of the fissure of Sylvius. To do this, draw a line, starting one and one-quarter inches posterior to and on a level with the external angular process of the frontal bone, to a point, three-quarters of an inch below the most prominent point on the parietal bone. This line marks the horizontal limb of the fissure. The ascending limb is given off at a point, three-quarters of an inch from the anterior extremity of this line. A base line is then drawn from the lowest portion of the infraorbital margin backwards through the external auditory meatus. From this he erects two perpendicular lines, extending to the median line, one passing through the depression in front of the external auditory meatus, and the other from the posterior border of the mastoid process at its root. The point of junction between this latter line and that marking the longitudinal fissure gives the upper extremity of the fissure of Rolando, while the lower extremity is indicated by the point at which the anterior of the vertical lines and the line marking the fissure of Sylvius intersect.

The occipito-parietal fissure is indicated by the point at which the line for the fissure of Sylvius, if prolonged, strikes the sagittal suture.

The sulcus between the first and second frontal convolutions lies beneath a line starting from the supraorbital notch, and running parallel with the line of the longitudinal fissure, and stopping two-fifths of an inch in front of the line indicating the position of the fissure of Rolando.



## GENERAL PARALYSIS OF THE INSANE.

**Synonyms.**—Paresis; paretic dementia; paralysie generale (French); allgemeine progressive Paralyse (German).

**Definition.**—General paralysis of the insane is an organic disease of the central nervous system, characterized clinically by ataxia of movement and general loss of motor power, and associated with mental deterioration, the latter being first manifested by simple changes in disposition, and later by most marked physical changes, mania, delusions generally of an expansive character, and, finally, dementia. Pathologically, the disease is characterized by organic changes in the cerebral cortex, the spinal cord and the sympathetic nervous system, the character of which will be explained shortly.

**History.**—The disease under consideration appears to be a creation of modern life, as descriptions of cases corresponding to general paralysis of the insane are not found in medical literature prior to the commencement of the present century.

**Etiology.**—The clinical investigation of general paralysis of the insane shows that in nearly every instance no one factor can be assigned as the cause of the trouble. It appears rather that the disease is the product of a variety of causes to which the patient has been exposed for years. The predisposition of men to the disease has always been noteworthy; indeed, it was believed for a long time that it never attacked women. Later experience, however, shows that in about one-sixth of all cases the subjects are women. Age, also, is a powerful predisposing factor, nearly all cases beginning between the ages of thirty-five and forty. The disease rarely commences before the age of thirty or after sixty. English authorities make the predisposing age somewhat greater than above given, *i. e.*, from forty to fifty years. The earlier age assigned by American authorities finds explanation probably in the great brain pressure and strain to which men of our nationality expose themselves.

Heredity has some bearing in the production of the disease. It is not a direct one, however; *i. e.*, the transmission of insanity in the parent to the child, but rather of the peculiar mental and physical traits and passions, the bent of which is to the production of general paralysis of the insane. From this standpoint, heredity may be said to be an etiological factor in about one-third of all cases.

Occupation has considerable influence in causing general paralysis of the insane. Those exposed to great heat and cold, as soldiers and certain artisans, have the disease with more than average frequency,

We also find this to be the case with members of the learned professions, medicine and law, the productive agent here being the great mental strain to which physicians and lawyers subject themselves. Many prominent actors and authors have also succumbed to this disease, especially has this been noted in those who have not only been subjected to prolonged mental strain, but have ruined themselves constitutionally by alcohol, venery and syphilis.

The upper classes of society, in whom an impressionable nervous system has been developed, seem to be especially subject to the disease. It has been noted that among them, women are attacked with more than the average frequency.

In some cases, head injury or sunstroke seems to be the starting point of the trouble.

Alcoholic indulgence and sexual excesses are found to be the most prominent of the exciting causes of the disease. Without doubt, unnatural sexual practices are etiological factors; yet it seems also that the frequent consorting with lewd women, with its attendant revelry, are more active.

In the opinion of most alienists, syphilis has but little influence in causing general paralysis. My own experience, obtained entirely outside of asylum practice, leads me to the opposite conclusion, for in nearly all cases coming under my observation, a clear syphilitic history was obtainable. I have always felt that asylum statistics bearing on this point are unreliable. Perusing the last report of one of our well-conducted large asylums, I note that syphilis is given as the cause of the insanity in but three cases. It is generally stated by alienists that there exists a "pseudo-paresis" of syphilitic origin, closely simulating the idiopathic disorder, and amenable to antisiphilitic measures. The general experience of practitioners, however, will show that these cases are by no means as amenable to treatment as are other syphilitic processes.

The mental causes of general paralysis of the insane are found in a life of ambition and competition, the subject constantly exposing himself to mental strain and anxiety and depriving himself of physical comforts and mental rest. It is not the activity of a short period that is dangerous, but the long-continued mental effort without variety, often associated with loss of sleep.

The above general review of the causes of general paralysis of the insane shows that no one factor is at the bottom of the trouble, but that many causes are at work ere the disease asserts itself.

**Pathology and Morbid Anatomy.**—It is universally believed that the primary pathological changes in general paralysis occur in the cerebral cortex and its investing membranes, and these are followed by others of similar character in the medulla and spinal cord. Exception-

ally the disease starts in the cord as locomotor ataxia, the cerebral changes appearing later. In still another class of cases the changes begin simultaneously in brain, medulla and cord. Cases having all the clinical features of general paralysis have been observed in consequence of gross morbid cerebral changes, as tumor. In these instances, the primary lesion undoubtedly sets up a chronic inflammation in the neighboring cortex.

The causes leading to the cortical degeneration of this disease, are undoubtedly multiple. Congestion probably plays an important part in many cases. Certainly, this condition can arise very readily from the mental strain and application which so frequently produces this disease.

Some hold that the changes in the brain are the result of a premature senility, in other words, an atrophy of brain structure in the prime of life. Still others teach that it is the nature of a chronic inflammation.

During the formative stages of the disease, it is probable that the intense mental application sets up a cortical hyperæmia, which, in its turn, reacts upon the brain-cells, maintaining them in a state of morbid activity. Long continuance of this condition results in vascular changes, and alterations in cerebral nutrition. The changes to be described shortly, ultimately result. The mental deterioration of the disease is the result of these changes invading the frontal lobes of the brain. The exalted delusions are attributable to the prolonged hyperæmia of the ideational centres. The depressed mental states have been explained by the alterations in the composition of the blood. The various physical phenomena arise from changes in the portion of the central nervous system subserving the function disturbed.

**Clinical Course.**—The onset of general paralysis of the insane is, with but few exceptions, a slow one. For descriptive purposes, the disease may be divided into three stages, the first of which is ordinarily called the stage of prodromic symptoms. This is characterized by departures from the normal behavior or rules of conduct on the part of the patient. He is observed to become more irritable than was his wont; he exhibits an emotional state of mind, and more or less lack of self-control. Ofttimes there appears a perversion of the moral sense. Sexual desire becomes abnormally strong, and this, taken in conjunction with the deficient self-control, sometimes leads to the most outrageous conduct even in public, and in the midst of the afflicted one's family. In some cases, the deficient moral sense is exhibited in a kleptomania; in others, by profanity, the use of obscene language, etc. The sexual powers are not always exalted, however, some cases showing the opposite condition, that of loss of both power and desire. Craving for alcohol, with increased sensibility to its effects, often occurs. All these changes in morals are liable to take place, irrespective of the high standard pursued by the



patient when in health. Very characteristic of the early stage of the disease is an exaggerated self-importance, which leads the patient into the most extravagant, even impossible, business schemes. In the pursuit of these wild plans, his legitimate business is generally sadly neglected. Motor defects are observed in the shape of inco-ordination of movements affecting muscles requiring a nice adjustment, as those of the tongue, lips, and fingers. Disorders of speech are common, and are manifested by stammering, dropping of syllables and words, and the use of the wrong words.

Sensory symptoms are often observed and consist of a variety of *anæsthesias*, even affecting the special senses. Neuralgia may attack any portion of the body, even the various viscera. Headache is by no means uncommon, and, when present, may be very severe.

The second stage of the disease is the first stage of the fully developed general paralysis. Usually the prodromic symptoms merge gradually into the second stage; occasionally, however, the latter is marked by the sudden onset of a mania, which is characterized by exaggerated ideas and delusions. The paretic symptoms already mentioned as found in the first stage are present, but in an intensified degree. Speech is even more visibly affected. Words containing linguals and labials give especial difficulty. The mania may be of such a violent type as to cause death by exhaustion.

In other cases, the patient retains his exalted delusions. He imagines himself the greatest architect, or the wealthiest man in the world. One patient seen by me a number of years ago, scorned to bother himself with such a puerile scheme as bridging the Delaware, as his time was taken up with plans to bridge the Atlantic Ocean. His general state of mind is one of self-satisfaction or egotism. He expresses himself as in the best of health. These delusions of exaltation in general paralysis, while very common, are by no means necessary to the disease, as they are absent in about one-half of the cases.

During the second stage, epileptiform and apoplectiform seizures are often observed.

The characteristic of the third stage is a gradually progressive dementia. Memory fails, especially for recent events. The gait becomes unsteady, oftentimes next to impossible. Speech becomes more and more defective, consisting of a drawl or even of an unintelligible jargon. The epileptiform seizures increase in frequency. The patient becomes more and more helpless and finally takes to his bed. Bedsores are now liable to form; gangrene of the lungs or pneumonia often sets in and death ensues.

The progress of the disease is often characterized by most remarkable remissions of symptoms, remissions sometimes so complete as to lead friends and family into the hope of a cure.

**Analysis of the Symptoms.**—**MENTAL CHANGES.** The mental changes of general paralysis have already been outlined as consisting of an exaltation and a general deterioration of all the faculties, especially, however, of memory and reasoning powers. Other peculiarities are present, and sometimes become so prominent as to obscure the true nature of the trouble. The dementia when well-marked is often accompanied by most careless and even filthy habits, together with deficiency in every direction from a mental standpoint. The delusions with exaltation have already been mentioned. For a long time, they were regarded as pathognomonic of this disease; but they are now known to be absent in some cases, and present in other forms of insanity than paresis. The direction taken by this exaltation varies greatly in different cases. In one patient, it is simply a state of self-satisfaction. He feels happy, regards himself as in the best of health, refuses positively to have anything to do with doctors or their treatment, while his whole manner indicates his depreciated mental state. Another class of patients is full of the wildest schemes for enriching themselves and humanity. Nothing seems to be too absurdly difficult for them to undertake. Some cases are characterized principally by mental excitement, during which they manifest a destructiveness and restlessness. Their conduct is often of the most indecent character imaginable, and that despite a previous life of propriety. Sometimes the mania is of rapid onset, and even carries off the patient from the incident exhaustion. Probably the most puzzling cases are those in which the mental condition simulates that of hypochondriasis. All delusions then centre on abnormalities of the different organs, principally, however, on the condition of the stomach and bowels. In another class of cases, the melancholic type of mental impairment prevails. The patient often has delusions relating to religion and persecution. Lastly, there are cases in which there is an alternation of excitement and depression.

**DISTURBANCES OF CONSCIOUSNESS.** The course of general paralysis of the insane is sometimes marked by attacks during which the patient becomes unconscious. The most prominent of these are the epileptiform and the apoplectiform seizures. The epileptiform convulsions may assume almost any character. They are sometimes the first phenomenon to direct attention to the patient's loss of health. Usually, however, they display some feature that distinguishes them from the convulsions of idiopathic epilepsy. Very often it is their duration, with the accompanying data suggestive of organic brain disease; in other cases, they are of the Jacksonian type; and in still others, they are unattended by loss of consciousness. Very often indeed, they are followed by impairment of motor power in some portion of the body. Sometimes they take the form of the status epilepticus, and thus cause death. The temperature is often elevated to a very high point as the result of their long continuance.

The apoplectiform seizures likewise assume several types. Sometimes they develop gradually in the course of one twenty-four hours, only to disappear as gradually during the next, leaving him with a certain amount of clumsiness or motor inco-ordination. Another variety of attack simulates the true apoplectic seizure very closely, and may end fatally. Some of these cases are followed by hemiplegia.

Sudden attacks of syncope mark the course of some cases of general paralysis. The seizures, as a rule, last for but a few minutes.

The MOTOR DEFECTS in general paralysis consist mainly of inco-ordination or ataxia of movements, especially of those requiring nice adjustment. The difficulties in speech have already been referred to. The writing will show the loss of power in the hand. Letters are formed awkwardly. As the result of the mental condition, words and syllables are omitted or misused.

PUPILLARY DISTURBANCES consist of inequality of the pupils, myosis, mydriasis, or the Argyll-Robertson pupil. They may be present in any stage of the disease, although more likely to appear in the fully developed periods.

The KNEE-JERKS may be either exaggerated, normal or diminished; or, again, one side may present alteration in one direction, while the other is very different.

The BONES undergo changes. Sometimes they become soft, while in others they grow abnormally brittle, and become liable to fracture from very slight strain, even from that of an unusual muscular exertion.

JOINT COMPLICATIONS, arthropathy as in tabes, mark the progress of some cases.

**Diagnosis.**—The recognition of general paralysis in its earliest stages is an important matter for the protection of both the patient and his family. The extent of misery and want to which the patient's financial schemes may bring him, can readily be understood, while his utter lack of moral sense may lead him to commit acts which bring disgrace forever to all connected with him by ties of blood. Unfortunately we cannot make positive statements as early as we would like. Many cases of neurasthenia and hypochondriasis bear some resemblance to general paralysis, and must be distinguished. For the recognition of the disease under consideration, we depend mainly on the alteration in character, the speech defects, the motor inco-ordination, and the pupillary changes. The sex and age of the patient, as well as his previous habits of living, are of considerable value.

*Alcoholic insanity* may be confounded with general paralysis. The tremor in the disease is, however, more general, and the disturbance of speech seems to be in accordance with the severity of this symptom. Ataxia is a less prominent symptom, and the delusions are of a suspicious type.



Those who hold that there is a distinction between general paralysis of the insane and syphilitic pseudo-paresis, give as data for their clinical differentiation the predominance of the physical over the mental phenomena in the syphilitic disease. Especially is the latter trouble suggested when the mental disorder has been preceded by palsy of any of the cranial nerves, especially, however, those going to the muscles of the eye. Other conditions suggestive of the syphilitic pseudo variety are the absence of the exalted delusions, the tremor of the lips and tongue, the more irregular course, and the better response to antisypilitic treatment.

*Acute mania* will be differentiated by the history of the case.

In *monomania* with delusions of exaltations, the delusions are of a fixed definite character.

**Prognosis.**—This is absolutely unfavorable, the incurability of the disease being generally acknowledged. The duration of the disease varies greatly, however. The average case ends fatally in about three years, and yet life may be prolonged much beyond this period. Many cases are interrupted in their course by periods of remission, remissions so nearly complete as to lead to most delusive hopes.

The termination may be from exhaustion or by the complication with some intercurrent disorder, as pneumonia.

In some few cases, the progress of the diseases is checked, and the patient lives on for years in a defective mental condition.

**Treatment.**—Prophylactic treatment must be conducted on the lines suggested by a study of the etiological factors at work in the production of the disease. These include attention to every detail of hygiene. The course of the fully developed disease may be greatly ameliorated by the adoption of rules of life in accordance with the conditions present at different times during the course of the complaint. Food, exercise, rest, out-door recreation, bathing, etc., must all be regulated. As a rule, the patient will get along better away from home and relatives and *in the care of a skilled nurse*.

Personal cleanliness is of the greatest importance, owing to the danger of such trophic phenomena as bedsores.

Cases presenting a syphilitic history should be thoroughly treated by the administration of iodide of potassium in large doses, as already indicated in the article on syphilis. While the results are not such as one would wish, they are oftentimes most excellent, either staying the course of the disease, or greatly ameliorating the condition of the patient. Mercury in any form is probably useless if prescribed as an antisypilitic measure.

The surgical treatment of general paralytics has attracted considerable attention, owing to reports of a few cases much improved after a simple trephining and opening of the dura, the idea being to reduce

intracranial tension by the operation. The general opinion of the best alienists is decidedly antagonistic to surgical interference, as being entirely useless.

As to remedies, *chloride of gold and sodium, platina, argentum nitricum, picric acid, secale, arsenicum, belladonna, hyoscyamus* and *stramonium*, are suggested as means of relieving intercurrent conditions.

Delusions in general paralysis, as in other insanities, present such variations that no article can provide for the management of all cases. Emergencies arise which call for the exercise of tact, and it is in this measure that the success in the treatment of this disease and many mental cases is obtained.

## ACUTE PERIPHERAL ENCEPHALITIS.

**Synonyms.**—Bell's mania; typhomania; acute delirium; grave delirium; phrenitis.

**Definition.**—An acute disease of the brain, characterized clinically by violent delirium and mania, with periods of intermission, and accompanied by anæsthesia of some portions of the body, and dependent pathologically upon hyperæmia or inflammation of the cerebral cortex and overlying membranes.

**Etiology.**—This disease affects both sexes, with about the same frequency; active adults seem to be attacked more frequently than adolescents and the aged. The exciting causes of the disease are found in alcoholic excesses, a life of anxiety and deprivation, the acute infectious diseases, traumatisms, and sunstroke. Cases arise also in the course of certain chronic disorders, *e.g.*, diseases of the bones of the skull, locomotor ataxia, and general paralysis of the insane.

**Pathology.**—The origin of this disease is involved in considerable obscurity. It is commonly held that the lesions start as hyperæmia of the cerebral cortex, followed by an œdematous infiltration or exudation into the pia and cells of the cortex. Small hæmorrhagic extravasations are often observed. Some authorities believe that the condition arises from a constitutional poison of unknown character.

**Symptomatology.**—The onset of acute delirium is always sudden. For a few days the disease may be preceded by prodromic symptoms, these as a rule taking the form of increased mental activity. In other cases, there exists a general restlessness, with disturbances of sleep of varied character. Still other cases have as the initial symptom an epileptiform convulsion or attack of unconsciousness.

The mania or delirium develops rapidly. The mental condition of the patient is one of great excitement, with incoherent speech, and destructive disposition. He makes strenuous efforts to escape from his attendants; indeed, he may inflict serious injuries on them in his attempts. His exertions are often so violent as to exhaust him, although during their continuance he often exhibits almost Herculean strength. The temperature is high, ranging from 101° to 106° F., its height apparently being proportioned to the intensity of the delirium. The fluctuations in temperature are, however, remarkable; at one time the maximum figure is observed, and before nightfall or by the next morning it is but little above the normal. In the terminal stage of the disease, when stupor is about to appear, the temperature may fall below the normal. From the very beginning, sleep is greatly disturbed, if not entirely



absent. All the symptoms are greatly aggravated at night. Food is refused; the tongue is dry, and the pulse rapid and feeble. The violent mania is often interrupted by perfectly lucid intervals.

With the advance of the disease to its second stage, the patient becomes quiet, and gradually lapses into coma, or muttering delirium, and with evidences of collapse.

**Diagnosis.**—Acute delirium is especially liable to be confounded with the violent delirium which ushers in some cases of pneumonia. Such cases, however, are especially liable to occur in children, while acute delirium is a disease of adult life. Physical examination of the chest shows the signs of pneumonia.

*Acute meningitis* presents some similarity to acute peri-encephalitis, but its course is marked by hyperæsthesia, stiffness of the muscles of the back of the neck, and the severe headache.

*Acute mania* is not so liable to be associated with high temperature. Some authorities teach that high temperature is diagnostic of acute peri-encephalitis.

**Prognosis.**—The prognosis in acute delirium is highly unfavorable, a very large majority of cases ending fatally. The danger seems to be in direct proportion to the severity of the delirium and insomnia. The duration of the disease ranges from two or three days to three weeks.

**Treatment.**—Locally, cold should be applied to the head in the shape of cold compresses, or the ice-bag. The patient should be kept in bed, and as much as possible without the enforcement of any undue physical restraint. The remedies of possible use are but few, and include the well-known and well-tried solanaceæ, belladonna, hyoscyamus, and stramonium. While the results from these are not all that we would wish for, they are nevertheless better than can be obtained from any other remedies, for which reason their use should be persisted in as long as the symptoms of the case correspond with those of the remedy. If a hypnotic is required, it should be either hyoscine hydrobromate, in doses ranging from one one-hundredth to one three-hundredth of a grain, every four to six hours, or chloral hydrate. The administration of either of these drugs calls for judgment, as already intimated in the remarks on hypnotics in the chapter on general considerations relating to diseases of the nervous system.

The diet of these patients should be as light as possible, and yet nutritious. Milk and good beef broths are probably the most suitable articles on which to feed the patient.

The nurse or nurses should be selected for their adaptability to the management of maniacal patients, and not because of their physical strength (although strength has its value) or their skill in cases of another character. Tact will often quiet a patient when brute strength fails.

# DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

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## DISEASES OF THE SPINAL MENINGES.

The disease of the spinal meninges to be considered are three, namely, inflammation, hæmorrhage, and tumors. Clinically, hæmorrhage and tumors of the meninges are inseparable from the same conditions affecting the cord itself; they will therefore be considered in conjunction with hæmorrhage and tumors of the cord, under the titles spinal hæmorrhage and spinal tumors, respectively.

### INFLAMMATION OF THE SPINAL MENINGES.

Inflammation of the spinal meninges or spinal meningitis is but rarely observed as a primary affection, if we except those cases of epidemic cerebro-spinal fever in which the brunt of the disorder is borne by the spinal membranes. Many classifications of spinal meningitis have been offered, but most of them are entirely useless from the clinician's standpoint, as their recognition is oftentimes, if not always, impossible in practice. Thus there have been described pachymeningitis externa and interna, and leptomeningitis; and each of these may be either acute or chronic. In the following pages I shall follow the classification of Gowers as being the only practical one. That authority considers only an external and an internal spinal meningitis. The former includes only cases of inflammation of the outer surface of the dura mater; and the latter all cases in which the process affects the inner surface of that membrane with or without involvement of the pia mater and arachnoid.

### EXTERNAL MENINGITIS.

**Etiology.**—External meningitis occurs almost entirely as a secondary affection, resulting generally from caries or syphilitic disease of the vertebræ. When thus originating, it is very apt to pursue a chronic course, although causes exciting the inflammation to acuteness may obtain. The disease likewise arises from deep sacral bedsores, in which case the inflammation is very frequently indeed widespread and acute. Examples of meningitis externa have been known to follow an ascending neuritis. As a primary affection it may occur in young cachectic individuals as the result of exposure to cold.

**Pathology and Morbid Anatomy.**—The inflammation is limited to the outer surface of the dura mater, and may or may not be accompanied by suppuration. According to its intensity will the dura present a simple reddened appearance with but slight deposits of lymph, or a highly inflamed condition with extensive formation of pus. In many cases secondary to vertebral caries, the inflammation is localized strictly to the seat of the primary lesion, and is associated with a great accumulation of inflammatory products between the dura and the walls of the bony canal. This deposit often develops into fibrous tissue, which may entirely surround the cord, and contracting, produce marked constriction of that structure. In other cases it is nodular in distribution. In primary meningitis externa the inflammation is widespread, and the suppuration not infrequently profuse. The pus may confine itself to the space between the dura and the bone, or it may burrow between the vertebral arches, and make its appearance on the surface of the back. Occasionally, it forces its way forwards, and appears anterior to the bodies of the vertebræ, in which case, pressure by it produces symptoms of a most mysterious nature, if the existence of vertebral disease with suppuration is unknown to the physician. For example, in the case of vertebral disease high up with escape of pus anteriorly, the œsophagus is encroached upon, and difficult deglutition follows.

The prevalent idea that the paraplegia accompanying Pott's disease results from the compression of the displaced bones, should not be entertained, for in nearly every instance the compression is exerted by the inflammatory effusion. Proof of this statement is found in the numerous examples in which the paraplegia occurs independently of any deformity, and of still others in which the deformity exists for years before any loss of power is manifested.

**Symptoms.**—The symptoms of external meningitis may be divided, into two classes: those dependent upon the primary disease, and those upon the meningitis *per se*. Cases will occur in which the latter class are so lacking in prominence that they fail to attract the attention they merit. I have long held, and experience has taught me the value of my opinion, that the slightest evidence of meningeal disease in connection with the symptoms of vertebral caries is not to be ignored. The presence or absence of spinal deformity does not affect the validity of this opinion in the least, for one will frequently find cases of external meningitis secondary to vertebral caries in which no deformity is present, and also others in which the paralysis disappears as the deformity becomes patent to observation. Cases of the latter kind find ready explanation. The breaking down of the bodies of the vertebræ serves to liberate the inflammatory products making spinal compression. Naturally under these circumstances, the function of the cord are in a measure restored.



In the majority of cases of external meningitis\* a history of injury producing the vertebral caries is invariably present. It is fixed to one particular spot, and is greatly aggravated by exercise, jarring and pressure, and is relieved by absolute rest. It is most marked in those portions of the spinal column permitting of most motion, hence in the cervical region. It is readily intensified by manipulation producing lateral movements of the vertebræ. In some few cases spinal caries is unattended by local pain. Careful palpation sometimes discovers a thickening of the tissues about the diseased bones; especially does this occur in the cervical region.

Probably the first evidence of intravertebral disease is found in increased reflexes. The patient sometimes complains of dull, aching pains in the extremities. The various superficial and deep reflexes dependent upon segments below the lesion exhibit all grades of exaggeration, from the mildest to the most extreme. The slightest tap on the patellar tendon is sufficient to excite a most energetic response. Ankle clonus is marked. The muscles of the back are more or less rigid. An exception to this is found in those cases in which the lumbar enlargement of the cord is severely injured by the compression. I have seen cases in which this exaggeration of the reflexes constituted almost the entire evidence of intraspinal disease.

The paraplegia is, in the majority of cases, of gradual onset. In some few cases it may reach its maximum severity in two or three weeks from its commencement; and, in still more exceptional instances, it has been known to come on suddenly in response to a slight traumatism, or without apparent cause, and reach its acme over night. Whatever be its onset, the paraplegia is always of the spastic variety. In extreme cases, the bladder and rectum may be paralyzed.

The acute cases of external meningitis are characterized symptomatically by local pain and tenderness, aggravated by any motion, especially such as cause rotation of the bodies of the vertebræ; by irritation of the spinal nerve-roots, as shown by local rigidities and spasms, eccentric pains, and hyperæsthesia of the cutaneous surfaces; anæsthesia and paralysis of the parts of the body supplied by the irritated or compressed nerves; increased reflexes; in bad cases, paralysis of the bladder and rectum, high fever and, when suppuration is at all profuse, complete paraplegia.

**Diagnosis.**—The association of spinal symptoms with a primary bone lesion is generally sufficient to establish the diagnosis of external meningitis. One of the greatest difficulties lies in the acceptance by physicians of the theory that the paraplegia accompanying vertebral

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\* Vertebral caries is by all odds the most frequent pathological cause of external meningitis, for which reason the disease as thus produced is mainly referred to in this description.

caries is entirely dependent on the deformity. "No deformity, no compression of the cord," think they. The fact as already stated is that the compression arises from the meningeal inflammation or the pressure of the inflammatory products arising therefrom.

External meningitis may be differentiated from *internal meningitis* by the recognition of symptoms pointing to the primary disease only. If this starts outside of the *dura mater*, then we are safe in diagnosing external meningitis.

External meningitis shows clinical differences from *myelitis*, in that it exhibits a higher degree of pain, and more spasm and irritation of the nerve-roots. Paralysis, moreover, is not an early symptom.

**Prognosis.**—Acute external meningitis runs an almost inevitably rapid and fatal course. Reported recoveries have been questioned. As to the chronic variety secondary to vertebral disease, the outlook is far more promising than in other spinal affections with equally unfavorable symptoms; even here the prognosis must be most guarded. The nature of the primary affection enters largely into the question of prognosis. Thus, if it be cancer of the vertebra, death must necessarily ensue shortly; if it be vertebral caries, then it depends to a great extent on our ability to control that destructive process. The outlook as to recovery from the paralysis depends upon the extent of damage done to the spinal cord itself. This can be gauged by the degree of anæsthesia in the paralyzed extremities. Still there will be many cases in which the paralysis fails to disappear entirely.

**Treatment.**—The treatment of external meningitis is essentially that of the primary disorders. Cases dependent upon vertebral caries, must be treated by the jacket, or absolute rest. *Silicea*, *fluoric acid*, *calcareæ*, *sulphur* and similar remedies may be thought of. The acute affection requires *belladonna*, *hepar*, *aconite* or *bryonia*. Syphilitic cases must be treated with *iodide of potassium* or *mercury*, or both.

When opportunity for giving vent to pus is afforded, it must be taken advantage of.

Cases dependent upon incurable conditions as malignant growths, call for palliative treatment, principally in the shape of morphia, hypodermically. The complications of external meningitis call for the treatment described in the chapter on myelitis, to which the reader is here referred.

## ACUTE AND CHRONIC INTERNAL SPINAL MENINGITIS.

### ACUTE INTERNAL SPINAL MENINGITIS.

Acute internal meningitis is an acute inflammation of the pia mater, arachnoid and inner surface of the dura. It may be limited strictly to the former membranes, or it may extend to the inner surface of the dura mater on the one hand, or to the spinal cord on the other. In the latter case it receives the designation of "meningo-myelitis." Inflammation of the pia and arachnoid alone is called lepto-meningitis.

**Etiology.**—Aside from extension of inflammation from the brain, acute internal meningitis may arise from local conditions or from morbid blood states. In the former category are included mechanical causes, as injuries, which may be either severe or slight, with or without perceptible injury to the spinal column itself, and exposure of the back to cold. Among the causes arising from morbid blood states are septicæmia, general exposure to cold, the constitutional infections of syphilis and tuberculosis, typhoid fever, and the peculiar blood state giving rise to rheumatic inflammation, and inflammation of serous membranes generally. Dana regards the rheumatic cases as probably arising from an unrecognized infection. Acute internal spinal meningitis is especially apt to occur in young men.

**Morbid Anatomy.**—The inflammation may be either simple, purulent or tuberculous. It is usually of wide distribution. In the earliest stages the membranes present but a slightly reddened appearance, with perhaps a few ecchymoses here and there. When, however, the disease is more advanced, we observe also thickening and opacity of the pia mater and arachnoid, together with exudation of grayish-yellow inflammatory products. The cerebro-spinal fluid is increased in quantity. It may or may not be turbid from flocculi or pus. The roots of the spinal nerves are likely to be involved by the inflammation, though it is perfectly possible for them to be covered by the exudate, and even actually buried in pus, without themselves being the seat of inflammation. It is also possible for the pia mater to entirely escape. Should recovery ensue, cicatrization takes place, the membranes remaining thickened and adherent. Permanent changes in the spinal cord itself may still remain, and these may give rise to local or widely diffused sclerosis, with the secondary degenerations.

**Symptoms.**—The symptoms of acute internal meningitis do not



differ materially from those of the external variety. The disease is ushered in by pain in the back, rigors, fever and malaise. The pulse may be fast or slow. The pain in the back is aggravated by motion and pressure. Severe paroxysmal eccentric pains from irritation of the nerve-roots, muscular spasm as exhibited by rigidity of the muscles of the back, trunk and extremities, hyperæsthesia of various portions of the cutaneous surface, early increase of the deep and cutaneous reflexes, retention of urine, and constipation. The muscular spasms may be sufficiently severe to produce opisthotonos or other fixed positions of the body, *e. g.*, rigid flexion of the limbs. When the upper portion of the cord is involved, dyspnœa and alterations in the pulse are common. As the disease progresses, paralysis is added to the above symptoms; and this may become complete. Next, sensation is lessened or even lost. The reflexes then become diminished and finally abolished. At this stage the greatest care must be taken lest bedsores form; the bowels and bladder also require attention, as there may be constipation and retention of urine and fæces. The symptoms must, of course, vary in distribution, according to the extent and location of the central inflammation. The mental condition is, as a rule, unimpaired. In fatal cases, coma and delirium may precede death.

Owing to the diminished tendency of the nerve-roots to become involved in suppurative cases, irritation symptoms are slight in that variety. Tuberculous cases are likewise less obtrusive in their symptomatology. They, moreover, rarely occur without some involvement of the cerebral meninges, or evidences of tuberculosis in other organs.

**Diagnosis.**—From *myelitis*, meningitis is distinguished by the comparatively slight degree of pain in the back in the former. Paralysis occurs early in the course of myelitis, while rigidity is either slight or absent.

*Meningeal hæmorrhage* may give rise to meningitis; but the onset of these cases is sufficient to make their true nature manifest at a glance.

From *rheumatism* it is differentiated by its widespread spasm and pain, the eccentric nerve pains, the exaggerated reflexes, and the retention of urine and fæces.

**Prognosis.**—The prognosis is unfavorable in all cases. Many terminate fatally within a few days; others drag along for two or three weeks, and then die, or proceed slowly to a more favorable issue. Cases surviving the first week have a good chance for recovery. Sometimes, because of the heart becoming involved in the general asthenia, death follows late in the disease. Other cases succumb to bedsores. Kidney disease occurs in other cases from retention of urine, and brings about a fatal issue. In a general way it may be said that the prospects for recovery are better in traumatic cases, and those arising from exposure to cold. Cases that recover are sometimes the victims of various disabili-

ties and deformities from contractures, and affections resulting from injury to the nerve-roots.

**Treatment.**—Absolute rest in bed is the most essential element. It is undesirable for the spine to be the lowest portion of the body; so when consistent with the patient's comfort the lateral decubitus should be adopted. The greatest care and judgment must be exercised to avoid the formation of bedsores, and to prevent cystitis. The latter evil is readily obviated by emptying the bladder regularly with strict observance of cleanliness. Hot poultices or ice-bags to the spine prove valuable in some cases. Some cases are benefited by lukewarm baths, which lessen the tendency to bedsores and cystitis.

*Aconite* is the remedy best adapted to the early stage to control the oncoming inflammation. *Ferrum phos.* is likewise called for early in the febrile stage, when the pulse is soft and full, and before exudation has taken place. Later, one must have recourse to remedies suited to inflammatory conditions of serous membranes, *bryonia* being especially valuable in this respect. This remedy is highly endorsed by Hughes. It is especially adapted to rheumatic cases. *Rhus* is likewise indicated in rheumatic cases, and also when the disease has arisen from exposure. *Apis* is called for in tubercular cases; *hepar*, when suppuration ensues. *Belladonna* is a valuable remedy when pains become a prominent feature.

*Causticum*, *hypericum*, *cuprum*, *veratrum viride*, *secale*, and *oxalic acid*, have been reported as used successfully in meningitis interna.

In those rare cases in which the above remedies fail to control the pain, morphia may be resorted to.

### CHRONIC INTERNAL MENINGITIS.

Chronic spinal meningitis is an exceedingly rare affection. This view is entirely different from that held some time ago, when many cases were regarded as of this character. Even at the present day there is an altogether too common tendency to diagnose this affection. Thorough examination will generally show such cases to be either primary lateral sclerosis, or, what is more likely, hysteria.

**Etiology.**—Most cases occur in adults. Some are the continuation of acute attacks. As exciting causes we have repeated and long-continued exposure to cold, and injuries. When traumatism is at the bottom of the difficulty, the symptoms usually make their appearance very shortly after the reception of the injury; and yet some time may elapse before the symptoms appear. Alcoholism, syphilis and tuberculosis are likewise causes. As a secondary affection it exists in all cases of compression of the spinal cord, and in many cases of myelitis, in which the peripheral layers of the cord are involved.

**Morbid Anatomy.**—The inflammation leads to adhesion between the arachnoid and the pia mater on the one hand, and the dura mater

on the other. The spinal fluid is turbid and increased in quantity. The membranes are thickened and opaque. Small calcareous plates are found in the arachnoid, especially when the lumbar region is affected.

**Symptoms.**—The symptoms of chronic internal meningitis bear a certain resemblance to the acute disease, and yet there are some important differences. The same pathological influences are active in symptom-making, namely, meningeal and nerve-root irritation and damage to the nerve-roots and the cord. The irritative phenomena are especially liable to be in abeyance, while all the symptoms are less obtrusive than in the acute affection. Pain in the back is present, and this is aggravated by pressure made *between* the spinous processes. It is generally accompanied by rigidity of the back-muscles. Eccentric pains in the area of distribution of the irritated nerves are to be noted. They are sharp, darting or rheumatoid in character. Evidence of irritation of motor branches, as exhibited in spasm, is mostly absent. Hyperæsthesia of the skin, sense of constriction, and dull, heavy feeling in the legs, are other symptoms. Paralysis is a late symptom in unfavorable cases. Sensation is then apt to be disturbed. Various phenomena of spinal cord disease appear, as shown by inco-ordination of gait, muscular atrophy, paralysis of sphincters, bedsores, etc. The symptoms run a very irregular course. They very often improve markedly under prolonged rest. By many the marked paralysis is held to be evidence of extension of the inflammation to the cord substance.

**Diagnosis.**—The diagnosis of chronic spinal meningitis rests partially on negative evidence, that is, the failure to establish the presence of some other affection. As affirmative evidence we have the history of early irritation of the spinal nerve-roots and their subsequent destruction. The greatest liability to error lies in the likelihood of confounding chronic meningitis with hysteria or spinal irritation. In the latter the spinal column is tender throughout, though with local spots of increased soreness which change from time to time; and there are no radiating pains. The patient gives an account of numerous neuralgiæ, the distribution of which is not consistent with the probable seat of maximum meningeal irritation. The surroundings of the patient will aid us in some cases in arriving at a correct conclusion.

Inflammation limited to the spinal nerve-roots may confuse occasionally. Here meningeal symptoms are absent, and the eccentric pains and other evidence of radicular irritation are strictly limited to the nerves especially involved.

**Prognosis.**—The prognosis is generally unfavorable. In severe cases life is endangered. The most favorable results are to be expected in cases arising from traumatism or syphilis. The condition of the general health of the patient has much to do with the ultimate result.

**Treatment.**—Absolute rest is required in every case. If possible,



the patient should lie prone, as thus there will be decidedly less tendency to congest the spine and its membranes. Counter-irritation, even to the extent of the actual cautery, has been strongly urged by some authors; but, in the majority of cases, it is entirely unnecessary. Still in cases failing to yield other carefully-planned treatment, resort to it should be had. Syphilitic cases require *iodide of potassium* or *mercury*, or both. In other cases, one of the remedies mentioned under the head of acute spinal meningitis or myelitis should be tried.

## ACUTE MYELITIS.

In the strictest meaning of the term, myelitis includes all inflammations involving the substance of the spinal cord. When used clinically, however, it is applied only to cases in which the inflammation is diffused—that is, limited to no one particular system or tract of that organ. When so limited the condition generally receives a special designation, as in the case of poliomyelitis anterior or inflammation of the anterior cornua.

Myelitis may be transverse, in which case the inflammation involves more or less of a segment of the cord; it may be disseminated when it attacks the cord in foci, which may be more or less widespread; it may be central, involving only the gray matter.

**Etiology.**—The most frequent causes of myelitis are exposure to cold, traumatism, muscular strains, acute infectious diseases, syphilis, and certain forms of toxæmia. Resulting from cold, it may follow a wetting, or lying on cold damp ground. When of traumatic origin, the spinal column usually gives some evidence of participation in the injury. On the other hand, one must not forget that the spinal cord may suffer severe inflammatory action, while its bony envelope escapes unscathed. I might even go further and say that occasionally we meet with a most malignant myelitis following an apparently slight traumatism. As of traumatisms, so of strains; myelitis may follow the latter whether they be severe or mild. The most severe myelitis I ever saw occurred in a boy, and resulted from what was believed to be a sprain of the back produced by the act of throwing a club into a chestnut tree. Among the acute infectious diseases that bear an etiological relation to myelitis are typhoid fever, smallpox, inflammatory rheumatism and puerperal fever. Syphilis is an unquestionable and frequent cause, and may be a predisposing factor in many cases considered traumatic. The myelitis may follow the specific infection within the space of a year, or it may be delayed for an indefinite period. Lead and arsenical poisoning have caused myelitis.

**Symptoms.**—The premonitory symptoms of myelitis are those of the acute inflammatory affections generally; high temperature,  $101^{\circ}$  to  $103^{\circ}$ , rapid pulse, associated with the usual constitutional phenomena. Convulsions are observed only in the case of children. The symptoms peculiar to the disease itself consist of a combination of motor and sensory phenomena, the former being as a rule the first to appear. The patient first experiences a sense of dragging or heaviness in the limbs, gradually

increasing in intensity until it amounts to absolute paralysis. Shortly after the onset of the motor weakness, sensory phenomena, consisting of numbness and formication, appear; and these increase more or less rapidly to complete anæsthesia. The time occupied by these symptoms in reaching their maximum intensity varies in different cases; it may be but a few hours in some, while in others it may not be for several days. In traumatic cases, when the cord has been greatly compressed, the motor and sensory paralysis is absolute from the start. The inflammation of the cord and its special symptoms are secondary.

Characteristic of myelitis, and nearly always present, is the girdle or cincture sensation. It is generally found at a point corresponding to the upper limit of the inflammation. Just above the seat of the girdle sensation is a band of hyperæsthesia. This latter phenomenon may be detected by the application of a test tube containing hot water to the part, and this will excite the pain as soon as it touches the hyperæsthetic area.

Pain in the back is scarcely ever a prominent symptom of uncomplicated myelitis.

The trophic phenomena of myelitis are most distressing. The skin usually suffers, becoming dry and harsh. The temperature of the paralyzed limbs is nearly always higher than that obtained under the tongue. The most important lesion, however, and the one for which the medical attendant must ever be watchful is the bed sore, a complication dreadful to witness, and most trying to the skill of the physician. Bedsores occur on all parts exposed to pressure, especially are they likely to appear over the buttocks and sacrum. Even the light pressure of the heels on the bedding frequently produces them in that locality, and I have even known them to attack the soft parts over the internal condyles of the femora from too constant adduction of the thighs. Bedsores may appear within a week of the onset of the attack, and destroy the soft parts with wonderful rapidity. While generally due to pressure, the latter condition is not a *sine qua non*. Draper has reported a case of gastric ulcer alleged to be the direct result of myelitis, and other observers have noted sloughing of the connective tissue about the bladder and rectum. Multiple arthritis has been occasionally met with.

The bladder and rectum rarely escape in acute myelitis. Usually, there is incontinence of both urine and fæces. The urine almost invariably has a highly ammoniacal odor.

When an entire transverse segment of the cord is involved, the reflexes depending upon the integrity of all segments below the one affected are destroyed. If, however, there are some fibres still intact, these reflexes are preserved, or even exaggerated. The muscles, receiving their nerve-supply from the anterior cornua in the affected segment undergo atrophy, and present the reaction of degeneration.



When the cervical portion of the cord is the seat of the pathological process, all four extremities are paralyzed. In addition, there will usually be observed irregularity of respiration. Optic neuritis is sometimes present. In one instance, exophthalmos resulted.

**Diagnosis.**—Acute myelitis may be confounded with acute poliomyelitis, Landry's paralysis, spinal hæmorrhage, multiple neuritis, hysterical paraplegia. In *acute poliomyelitis*, sensation is undisturbed; sensory symptoms are entirely absent; the deep and superficial reflexes are destroyed, the bladder and rectum are never involved; there is no tendency to bedsores, and the reaction of degeneration is present.

*Spinal hæmorrhage* is of sudden onset, and is attended with acute pain. The subsequent phenomena are generally the same as myelitis, for inflammation is very liable to follow.

In *acute ascending*, or *Landry's paralysis*, sensory symptoms are absent; there are no trophic phenomena; and the electrical reactions are normal; the functions of the bladder and rectum are unimpaired; then, too, the paralysis is of a progressive type, as its name signifies, beginning in the lower extremities, and gradually ascending.

*Multiple neuritis* has pains along the course of the affected nerves; the bladder and rectum are unaffected. The paralysis and anæsthesia are distributed according to the nerves affected. Bedsores are absent.

In *hysterical paraplegia*, there are no muscular atrophy, no bedsores, and no vesical or rectal incontinence.

**Prognosis.**—This is nearly always unfavorable, very few cases recovering. When life is prolonged for more than a few weeks (a fatal issue may result within three or four days), exhaustion, as a result of bedsores or renal disease, may follow, and eventually kill. Other cases develop into the chronic form of myelitis and last indefinitely. Some few cases exhibit a tendency to recovery.

**Treatment.**—The first great point in the treatment is rest of the most absolute kind. Then every precaution must be adopted for the prevention of bedsores. With this end in view, a water bed should be procured whenever possible. The bedclothing must be kept clean and free from wrinkles. The patient should be kept scrupulously clean and dry. The patient should not be maintained too long in one position, as it is desirable to frequently shift pressure from one part to another. When bedsores appear they should not be superstitiously regarded as some mysterious dynamic entity, and some absurd old-woman's treatment adopted. On the contrary, a common sense view of the situation should prevail, and the treatment conducted on strict surgical principles. If catheterization becomes necessary, careful aseptic precautions must be adopted. As local applications for the prevention of bedsores weak solutions of tannin or alum and plain alcohol may be recommended.

The selection of remedies is to be guided oftentimes by the nature of

the exciting cause, or the diathetic condition present. Traumatic cases require *arnica*, *aconite*, or *hypericum*.

Those arising from exposure to cold or damp require *rhys* or *dulcamara*.

*Cuprum ars.* and *arsenic* have been known to produce typical cases of myelitis, and should be administered in the uncomplicated cases.

*Mercurius* and *kali hyd.* singly or in combination, are the favorite old school remedies in myelitis. As shown by Baehr and Kafka, they are perfectly homœopathic to the peculiar character of inflammation present. It is not necessary to restrict their dosage to the potencies. If necessary, there should be no hesitation in giving the crude drugs.

Mercury has all the phases of the progressing paralysis of the extremities, of the bladder and rectum, with tendency to shocks and twitching, spinal pains aggravated by pressure, anæsthesia.

In the latter stages of the disease, when it tends to pursue a chronic course, *strychnia* is strongly advocated by Hale. There is every reason to expect considerable from this drug. Following Hale's rule of dosage, it should be given in a crude form, one sixtieth of a grain three times daily. Jewell, a prominent old-school neurologist, has likewise advocated the use of *strychnia* in myelitis, for which action he has met with considerable criticism from his colleagues.

Electricity may be used after the acute symptoms have subsided. Rockwell has reported one case of recovery from transverse myelitis by spinal galvanization without resort to other measures.

The continuous current was first applied to the spine. Then sharp and strong interruptions were practised in order to re-awaken irritability. Applications were also made to the muscles.

## CHRONIC MYELITIS.

**Etiology.**—Chronic myelitis may be observed as the continuation of the acute form, or it may exhibit chronicity from the inception of the disorder. In the latter case, it is especially apt to follow repeated exposure to damp and cold or injuries. The latter usually acts to produce a small lesion, which at first may be insufficient of itself to cause symptoms, and this subsequently excites a slow inflammatory process about it. Chronic alcoholism, the gouty diathesis, and syphilis are likewise causes.

**Symptoms.**—The onset of chronic myelitis is very gradual. Owing to the fact that the inflammation involves any or all of the different systems or structures of the cord, and at any level, the symptoms must present the greatest variety in different cases. Indeed, they may resemble those of almost any chronic spinal affection. Motor disturbances are generally the first to appear. They are manifested by a weakness of the lower extremities, which slowly increases from a paresis until it is worthy

of the designation paralysis. The affected limbs are usually more or less rigid. Sensory impairment likewise occurs, and this too increases slowly to more or less complete anæsthesia. The reflexes, both deep and superficial, are exaggerated. The bladder and rectum are affected, as shown by difficulty in emptying the bladder and constipation on the one hand, and incontinence of urine and feces on the other. There may be atrophy of some of the paralyzed muscles. When the disease has become well advanced and the patient is helpless, bedsores and cystitis may develop. The general health is usually preserved as long as the patient is able to go about.

**Diagnosis.**—As already indicated, chronic myelitis bears resemblances symptomatically to almost every chronic affection of the spinal cord. Its diagnosis should rest on the development of symptoms indicating changes in the cord, developing at random so to speak; that is to say, a lesion involving several systems of the cord. It resembles primary lateral sclerosis more closely than any other affection, from which it may be differentiated by the absence of sensory impairment in the latter affection. Tumor of the cord may present a close similarity to chronic myelitis; but it is accompanied by considerable irritation of the spinal nerve-roots.

**Prognosis.**—Chronic myelitis runs a course ranging from three to fifteen or more years. It may be regarded as incurable. Yet proper treatment will in many instances stay its onward progress, and give the patient a new lease on life. Cases arising from compression often exhibit a wonderful capacity for repair, when the exciting cause has been removed.



## ACUTE POLIOMYELITIS ANTERIOR.

Acute poliomyelitis anterior is a disease characterized at its onset by fever of greater or less severity, associated in some cases with convulsions and other morbid cerebral phenomena, and followed by a rapidly appearing paralysis. This paralysis generally assumes the form of paraplegia, and is more marked in some of the invaded parts than in others. It is not accompanied by any derangement of sensation. After reaching its acme, the palsy remains stationary for a time, and then undergoes a more or less marked regression.

It is also spoken of as acute atrophic paralysis, acute spinal paralysis, regressive paralysis, and infantile spinal paralysis. Owing to a mistaken idea of its pathology, it was known for many years as the essential paralysis of children.

**Etiology.**—The etiology of acute poliomyelitis is largely a matter of conjecture. Heredity, dentition, injuries, cold, and acute diseases, have all been assigned as causes, but with insufficient reason. In but few cases can a neuropathic family history be traced. Still some authors consider a neurotic constitution a very important predisposing factor. Dentition seems to have been dragged in as an etiological factor simply because it has been a prevalent custom for many years to attribute to it all obscure disorders at that period. Very few cases are known to have followed traumatism, and even in these few the relation of cause and effect has not been made clear. Cold can certainly have but little to do in producing the disease among children, for the vast majority of cases come on during the hot summer months. In adults cold does seem to be an etiological factor. Loweran has observed that this disease is of comparatively frequent occurrence among soldiers, probably because of the exposures to which they are subjected. While acute diseases apparently act as an occasional cause, yet a careful investigation of many of the cases alleged to have thus arisen, will show that the so-called acute disease was in reality but the primary fever with which most cases of acute poliomyelitis are ushered in. Strümpell even takes the ground that the paralyzes occasionally observed after measles, scarlatina, small-pox and other infectious diseases, although of spinal origin, are not of the same nature as the acute spinal paralysis of children. It is not necessary that the victim of this disorder be in poor health, for it attacks the strong and vigorous as well as the weak and puny. Inherited diathetic states exert no etiological predisposition. It has been known to attack patients during the lying-in period.

Age is a powerful predisposing factor. The majority of all cases occur in young children, especially in those between the ages of one and four years. It may occur, however, at any age. It is the predisposition of children to acute poliomyelitis that led to its name "acute spinal paralysis of children."

There is a growing conviction that acute poliomyelitis is dependent upon an acute toxæmia, the poison expending its chief action upon the anterior horns of the spinal cord. The evidence in favor of such a theory is largely of a hypothetical character, no positive evidence in its favor having been advanced. Epidemics of acute poliomyelitis may occur, but they are very rare. Thus Medin, of Stockholm, reports one in which he observed forty-four cases in the space of five months. Marie is a very ardent advocate of the infectious origin of acute poliomyelitis. He finds a strong reason for his belief in the frequency with which the disease occurs after the acute infectious fevers. While this may have been the result of his own experience, that of the majority of clinicians tends to show that poliomyelitis is of but exceptional occurrence at such times. Marie's suggestion, that vascular degeneration following acute infectious diseases is a cause of acute poliomyelitis, is well worth bearing in mind.

**Symptomatology.**—As there are some important distinctions between the symptoms of the disease as it attacks children, and those when adults are the victims, it is well for descriptive purposes to treat of its symptomatology under two heads: (1) Acute poliomyelitis of children; and (2) Acute poliomyelitis anterior of adults.

**Acute Poliomyelitis Anterior of Children.**—As is the case with the majority of diseases common among children, acute poliomyelitis is usually ushered in by fever together with such symptoms as general malaise, mental irritability, headache, and other evidence of cerebral irritation. The fever itself is rarely severe, though it may according to Strümpell rise as high as 106° F. It may be absent in exceptional cases or be unnoticed because of its appearance in a single night. Its usual duration is from twelve to forty-eight hours, but it has been known to persist for fourteen days. There is no fixed relation between the severity of the fever and the extent of the paralysis.

Occasionally convulsions constitute the first symptom of acute poliomyelitis. They are generally limited to the extremities, and rarely involve the face. They, together with the other cerebral symptoms, are probably the result of the general infection which causes this disease.

In rare instances the disease may assert itself without any prodromic symptoms whatever. In the evening the patient may have been put to bed in possession of apparently good health, and yet in the morning one or more limbs are found to be paralyzed.

The paralysis is always rapid in its onset, that is to say, it usually

reaches its acme within twelve or twenty-four hours from the inception of the first paralytic phenomena, after which it remains stationary for a time, and then undergoes a marked regression. Disorders of general sensibility are absent. Both the superficial and the deep reflexes of the affected extremities are totally abolished. The sphincters are hardly ever affected, we might say never, if we exclude the temporary incontinence of urine noted in some small children during the first twenty-four hours of the ailment.

At a period ranging in different cases from the fourth day to the end of the second week, alterations in the electrical reactions of the paralyzed muscles will be noted. These altered reactions have received the name of the "reaction of degeneration," and have been freely described in the chapter on general symptomatology of nervous diseases.

Shortly after the onset of the paralysis, sometimes as early as the end of the first week, the affected muscles undergo a marked and rapid atrophy, which is much greater than that which naturally follows the enforced non-use of the part. It may proceed in extreme cases until at last there is apparently no muscular tissue intervening between the skin and the bone. In some cases, the outward appearance of the limb does not give an adequate idea of the extent of the atrophy, because of the deposit of fat within and about the affected muscles.

Still later, the skin itself gives evidence of the malnutrition of the paralyzed extremities. It becomes cold and clammy, presenting a bluish or mottled appearance. It retains for a long time the impressions made by the patient's garters and stockings. The surface temperature may be 10° or 15° F. below the normal. The affected extremities are liable to frequent attacks of chilblains.

In no disease may the paralysis be of more variable extent than in the one under consideration. It may involve all four extremities together with the muscles of the trunk, or but part of one extremity only. In the latter case, the lesion in the spinal cord occupies but a circumscribed area, and is probably not of a severe grade. The paralysis may assume either the hemiplegic or paraplegic type, or it may invade the upper extremity of one side and the lower of the other. Usually, both lower extremities are affected, one of them, however, to a much greater extent than the other.

The improvement in the paralysis in mild cases begins within a week of the onset. According to the severity of the case, it may be delayed for several weeks. At the end of three months, most of the possible improvement will have been attained. Still, this should not lead us to relax our efforts for the patient's welfare, for persistent treatment for a year or more at least will still further increase the functions of the paralyzed part.

An arrested development of the bones is a common sequel of the



paralysis; and this arrested development is not necessarily proportionate to the degree of the associated muscular atrophy. In some cases, in which but two or three muscles are affected, and they but slightly, the nutrition of the osseous structures may be so greatly impaired that the affected limb will be several inches shorter than its fellow; and this shortening is further increased by the subsequent contracture. In other cases attended with extreme muscular atrophy and widespread paralysis, osseous growth proceeds uninterruptedly. The articular surfaces of the bones and the cartilages do not develop properly; and this, in conjunction with the lack of support to the joint, due to the paralyzed and atrophied muscles and relaxed ligaments, produces joint deformities, which sometimes exist to such a degree as to make the articulation capable of assuming most pitiable positions. The deformities which occur the most frequently, however, are those which result from contracture of muscles with but partially impaired function, and which are unantagonized by the natural tonus of their paralyzed and atrophied opponents.

**Acute Spinal Paralysis of Adults**, like that of infants, is ushered in with fever, malaise, headache, backache, etc., but rarely, if ever, with the convulsions which occasionally appear in infantile patients. The paralysis itself is more or less rapid in its appearance, and is associated with a diminution or abolition of reflex action in the affected extremities. The fever and other general symptoms soon pass away, and the paralysis begins to improve in those muscles which have not been seriously affected; while others, less fortunate, remain paralyzed and undergo rapid atrophy. The bones having reached their full development are not affected in this process. The skin itself is cold, relaxed and flabby. Deformities may occur in severe cases, but never to the extent seen in infantile spinal palsy. The electrical reactions are the same as those observed in the acute poliomyelitis of infants.

**Pathology.**—Owing to the infrequency of fatal results in the early stages of acute poliomyelitis, post-mortem examinations showing the condition of the spinal cord at that period are rare. For many years it was believed that the primary lesion resided in the muscles. It was Heine who first discovered the changes in the anterior cornua. These changes are briefly as follows: Examinations of the cord made within a few months after the appearance of the paralysis disclose a more or less diffuse inflammatory softening in the anterior cornua, and especially is this marked in the lumbar and cervical enlargements. (In the cases reported by Drummond and Turner the changes observed were those of an acute hæmorrhagic myelitis.) The majority of the large multipolar ganglion cells in these localities have disappeared, while those remaining are more or less atrophied. The nerve fibres and axis cylinders coming from the same have likewise been destroyed. The antero-lateral columns,

as a rule, are normal; occasionally they may be found sclerosed. The anterior nerve-roots are atrophied. In severe cases of long standing the changes in the spinal cord may be seen by the naked eye. Then shrivelling of the anterior cornua and, perhaps, of the antero-lateral columns is observed. At this stage, these areas are found under the microscope to be rich in connective tissue, while the ganglion cells and the nerve fibres are destroyed. This affection of the antero-lateral columns probably explains a phenomenon of this disease with which I have met several times, namely, that the knee-jerks, when restored, are much more vigorous and more readily excited than in health.

**Diagnosis.**—If the symptomatology of acute poliomyelitis, the fever, the rapidly-appearing paralysis, the reaction of degeneration, the marked muscular atrophy, the absence of sensory disorders, and the unimpaired functions of the sphincters be borne in mind, there need be no fear of confusing it with any other disease. The greatest source of error arises from the failure on the part of the physician to remember that other forms of paralysis occur in childhood, a lapse of memory that leads him to diagnosticate every paralytic trouble in infancy and childhood, acute poliomyelitis. Some little difficulty may be found in cases in which the incidence of the disease was not noticed by the parents. Under such circumstance, attention is not usually called to the trouble, until the time for the child to begin to walk has arrived. There may be danger, then, of confusing it with the *pseudo-paraplegia of rachitis*. In the latter condition, there are the characteristic, rachitic symptoms—beading of the ribs, enlarged and painful joints, and hyperæsthesia of the extremities; movements are possible, but painful, there is no muscular atrophy, or altered electrical reaction; diffused sweats are present; and the deep and superficial reflexes are normal.

From *spastic spinal paralysis*, it is to be distinguished by the reaction of degeneration, the muscular atrophy, and the remarkably relaxed condition of the paralyzed muscles. The two diseases, moreover, present widely different clinical histories, spastic spinal paralysis being of slow onset, and its phenomena progressive; while acute poliomyelitis is of rapid onset, and its symptoms regressive.

The most pardonable diagnostic errors are those in which *paralysis from nerve injury* is confounded with the disease under consideration. We need only the history of the case and the distribution of the paralysis to guide us. Paralysis may occur in infants as a result of tight bandaging, injuries during parturition, etc. Sensory impairment is the rule in these cases, and may be readily discovered in the case of adults. The paralysis is found to be limited to muscles supplied by certain nerve trunks. Complete recovery is the rule.

It is possible for syphilitic spinal cord disease to so closely simulate the acute poliomyelitis of adults, as to make a differentiation impossible. Such cases are rare, but, that they may occur, I know from experience.

**Prognosis.**—As regards life, the prognosis is favorable, death rarely occurring. It has been claimed that certain cases of sudden death in infancy are the result of acute poliomyelitis in which the nuclear cells in the medulla are attacked. As to recovery of the functions of the paralyzed parts, the outlook is not so favorable. On the contrary, complete recovery is rarely observed. Usually, one extremity regains nearly its wonted power, the other remaining more or less paralyzed. In the study of the electrical reactions of the paralyzed muscles, we have a ready means of deciding the future of any given case. When faradic contractility of the paralyzed muscles is lost early, that is, within a week after the onset of the disease, the paralysis may be regarded as permanent. If, however, the faradic irritability is retained for two weeks, or longer, a favorable result may be anticipated. According as faradic irritability is sooner or later lost, or more or less impaired, so may we prophesy as to the extent of the permanent injury.

**Treatment.**—At the very outset of the disorder, absolute rest should be enjoined, and *this rest should not be interfered with by ill-judged applications of faradism to the affected muscles for therapeutic purposes.* The paralyzed limbs should be well wrapped in cotton. Theory would seem to show the wisdom of maintaining the child in a recumbent position on its side or on its face, as such posture would tend to lessen the flow of blood to the spine. At the end of the first week, it will be well to test thoroughly the reactions of the affected muscles to both galvanic and faradic currents, *but this for diagnostic and prognostic purposes only.* Although at this time peripheral electrization is at least useless, if not harmful, galvanization of the affected area in the spinal cord will prove beneficial, if a mild and continued current be used. A large well-moistened sponge electrode attached to the positive pole is applied over the cervical or lumbar enlargement or both, according to the region affected, while the negative electrode is applied to some indifferent part, probably best on the anterior surface of the body. The sittings should be for five minutes and should be repeated daily. Along with rest and galvanism, the proper internal remedy should be administered.

*Aconite* is at once suggested because of the initial fever. It will, however, have lost its usefulness when the appearance of the paralysis announces the localization of the inflammation. Then we must look to *belladonna* or *gelsemium*.

The latter remedy has been highly recommended as the best in the early stages of acute poliomyelitis. Its provings show it capable of producing motor without sensory paralysis. It is also a valuable remedy in the early stages of infectious febrile diseases, of which acute poliomyelitis is possibly one.

When, after the paralysis has appeared the fever and other symptoms of inflammatory action continue, *belladonna* will be the remedy.



The higher the degree of local inflammation, the more sudden the paralysis, the more certainly will it be indicated.

After the stage of regression has commenced judicious applications of electricity to the paralyzed muscles will be of value. In those cases in which the muscles fail to respond to the faradic current, that current will be of no avail, and galvanism must be employed. The current selected should be of sufficient strength to produce muscular contractions and no stronger. The positive pole should be placed on the spine over the diseased area in the cord, and the negative over the motor points of the paralyzed muscles in turn. In fat subjects and in restless infants this may be impossible, in which case the positive electrode may be placed as above, while the negative is stroked over the paralyzed limbs. Care should be taken in making these applications that the muscles are not equally stimulated. Those muscles whose spinal centres have borne the brunt of the disease are the ones which should receive the greatest attention, while those which are but slightly affected require little or no electrical treatment. The sittings should be short at first, so as not to fatigue the weakened muscles.

At the time these applications are commenced, measures should be adopted to preserve the warmth of the limbs. Twice daily they should be bathed for fifteen minutes in water as hot as can be comfortably borne. Regular friction and massage of the paralyzed muscles must be practised daily. The inunction of cod-liver oil with the massage is a great aid to nutrition, and should be practised regularly. Passive movements of the limbs are necessary to prevent deformities. The limbs should not be permitted to remain in a dependent position, as this must interfere with the proper circulation of blood through them. Instead, they should be maintained as far as possible in a horizontal or elevated posture. Discretion in the selection of proper clothing is necessary; either spun-silk or wool should be worn next the skin. Before clothing the limbs, care should be taken that they are well warmed by artificial heat.

If the patient is old enough, and if he has recovered sufficiently, moderate exercise should be recommended, providing there is no deformity of the limbs resulting from the unequal distribution of the paralysis. For if such be the case, exercise will strengthen both slightly and severely paralyzed muscles alike, perhaps the former more than the latter, and so intensify the deformity. Before permitting exercise, then, see that all deformities are corrected, either by proper apparatus or by surgical operation. By a little ingenuity gymnastic apparatus for the exercising of individual muscles or muscle groups may be devised, and used with good effect.

. In the late stages of the disease, when the principal symptom is the paralysis with atrophy and deformity, *plumbum* is the remedy on which we should mainly rely.

*Graphites* may be suggested as an empirical remedy, on account of its influence over scar tissue. It should tend to lessen the cicatricial deposit in the delicate spinal structure.

*Causticum* is likewise a valuable remedy which has yielded good results in practice.

**Subacute Poliomyelitis Anterior.**—Under this head has been described a class of cases presenting the same symptoms as those already mentioned in acute poliomyelitis, but with a subacute onset. Unquestionably many of the cases brought under this designation are in reality examples of multiple neuritis. Others follow very strictly the course of the acute disorder with the exception of the rapid onset, and after a variable length of time, exhibit a natural tendency to recovery. Still others are steadily progressive from the beginning, and offer an unfavorable prognosis.

## SPINAL HÆMORRHAGE.

Hæmorrhage into the spinal canal may take place between the dura and the vertebra (extra-dural hæmorrhage), between the dura and the arachnoid (meningeal hæmorrhage), and into the substance of the cord itself (intra-medullary hæmorrhage).

**Etiology.**—Spinal hæmorrhage is in the majority of cases of traumatic origin; especially is this true of the extra-dural and the meningeal varieties. It may also occur from morbid blood states, as those exciting purpura, and following the severe infectious diseases, and disease of the vascular walls. It is a rare sequel of convulsive affections as tetanus and chorea. As a secondary condition it may complicate myelitis and spinal tumor. Meningeal hæmorrhage has followed the bursting of an aortic aneurism which had previously eroded the bodies of the vertebra, and rupture of an aneurism of one of the vertebral arteries.

Primary hæmorrhage into the spinal cord itself is very rare; indeed, the possibility of its occurrence has been denied. That it may happen cannot be doubted. It may affect patients of any age, but is most common during the first half of adult life. It may constitute part of a general hæmorrhagic tendency. Medullary hæmorrhage as a secondary affection occurs especially in connection with tumors and cavities in the spinal cord.

**Pathology.**—When due to a rupture of a bloodvessel it is usually because of fatty degeneration of its coats. Miliary aneurisms such as are so frequently observed in the brain are practically never found. In some cases the hæmorrhage precedes, or is the actual cause of, an attack of myelitis. The hæmorrhage may be single or multiple. In the case of extra-dural or meningeal hæmorrhage, the effused blood may spread itself throughout the entire spinal canal.

**Symptoms.**—The symptoms marking the onset of spinal hæmorrhage are more or less sudden. A severe pain in the back is experienced, and this is accompanied by numbness, tingling and formication. In case the meninges only are the seat of lesion, symptoms of irritation are more prominent, and hyperæsthesia of the cutaneous surface, and spasm of the back muscles appear. Finally paralysis with complete anæsthesia comes on. The sphincters are frequently affected. In the case of intra-medullary hæmorrhage, the symptoms of irritation are less marked, while the paralysis and anæsthesia are decidedly more pronounced. Involvement of the sphincters and formation of bedsores is a more prominent feature of hæmorrhage into the substance of the cord itself.



**Prognosis.**—This is unfavorable in all cases, especially when the cord is the seat of the injury. In cases which do not shortly result fatally, there is apt to remain a permanent paralysis.

**Treatment.**—Traumatic cases must be treated entirely on surgical principles. Cases which are followed by local inflammatory disturbance must be managed as described in the section on myelitis. It is well in all cases to apply an ice bag to the spine.

Among remedies, one should bear in mind *aconite*, *arnica*, *hamamelis*, and *veratrum viride*.

## TUMORS OF THE SPINAL CORD AND ITS COVERINGS.

Tumors of the spinal cord and its coverings are rarely observed. But little can be said of their etiology, excepting that which relates to the causes of tumors in general. No time of life is exempt from them, though they occur with greatest frequency in patients ranging from thirty to fifty years of age. In very many instances, they are secondary to growths of similar character occurring in other portions of the body.

**Pathology and Morbid Anatomy.**—Tumors may invade either the vertebræ, the meninges, or the cord itself. Parasitic growths, when intra-spinal, are not infrequently situated between the dura and the bone. The majority of spinal tumors find their origin in the meninges. Their next most frequent site is in the cord itself; and they occur the least frequently of all in the vertebræ. As a rule they are of small size; gliomata and sarcomata may, however, extend quite a distance in the direction of the longitudinal axis of the cord. The varieties of growths most frequently encountered are gummata, fibromata, sarcomata, and tuberculomata. The latter variety is very liable, according to the studies of Herter, to attack patients of less than thirty-five years of age. It is also associated with tubercular lesions elsewhere, mainly in the lungs.

Acting as a foreign body within the spinal canal, the tumor produces compression of the cord. The damage thus done will correspond with the size of the tumor and the rapidity of its growth. In the case of small and slowly growing tumors the cord oftentimes adapts itself to the new conditions. In larger growths, local degeneration ensues, and this extends along the cord, upwards or downwards, according to the direction of conduction of the tracts affected. Sometimes the progress of the tumor is characterized by attacks of inflammation, involving the meninges or the cord, or both. Some forms of tumor tend to the formation of cavities in the spinal cord.

**Symptomatology.**—It can be readily understood that the symptoms of spinal tumor must present great variety, owing to the many portions of the cord which may be invaded. Thus it is possible for them to bear most striking resemblances to the clinical phenomena of any of the system diseases of the cord already described in this work. In a general way it may be stated that the symptoms of spinal tumor are those of a focal lesion, which in its early stages produces phenomena indicating central irritation, and, subsequently, others showing compression and destruction of a transverse segment of the cord and

its nerve-roots. Of the early irritative indications, pain is prominent. It is apt to appear the earlier and be the more severe, as the tumor involves or approaches the meninges. While often experienced locally at the seat of the growth, it is more frequently found in the areas of distribution of the irritated nerves. Girdle sensation, anæsthesia, and hyperæsthesia, are also prominent symptoms. The situation of the paralysis will depend upon the location of the tumor. It almost invariably begins in one lower extremity. When situated in or above the cervical enlargement, it commences in the arm and leg on the corresponding side. Sensation is impaired or lost on the opposite side. The paralyzed muscles supplied by the nerve-roots given off from the affected segment of the cord, undergo atrophy and exhibit the reaction of degeneration. The reflexes from this segment are diminished or abolished. Those derived from lower portions of the cord are exaggerated.

The clinical course of a spinal tumor is sometimes marked by attacks of myelitis or meningitis or both, in which case the symptoms assume the character already described when treating of those diseases.

Tumor of the vertebræ is characterized especially by the severity of the attendant pains,—pains so severe as to lead to the special designation, *paraplegia dolorosa*.

**Diagnosis.**—The principal source of diagnostic error lies in the danger of confusing spinal tumor with the pachymeningitis of Pott's disease and cervical hypertrophic meningitis. Cervical hypertrophic meningitis is capable of acting mechanically as does a tumor; but its symptoms are bilateral from the start. The same is true in less degree of the paraplegia of Pott's disease. In the last-named affection, moreover, the radiating pains are not so severe as in spinal tumor.

**Prognosis.**—The prognosis of tumors of the spinal cord is exceedingly unfavorable, excepting in cases of syphilitic origin. The usual duration of the disease is from two to three years.

**Treatment.**—Syphilitic cases must be treated by measures usually employed in the treatment of syphilitic diseases of the nervous system. All other varieties of tumor can be cured only by surgical measures. When possible the tumor should be removed. It must be remembered that this operation involves profound shock and considerable danger. When the growth is so situated as to make its removal out of the question, laminectomy by affording the tumor greater opportunity for enlargement in a direction away from the cord, is a measure of considerable promise. The medical treatment of these cases must be conducted on purely symptomatic indications, the patient to be made comfortable at all hazards.



## ACUTE ASCENDING PARALYSIS.

**Synonym.**—Landry's paralysis.

**Definition.**—This disease, first described by Landry in 1859, is characterized by paralysis beginning in the lower extremities, and rapidly extending, involving in succession the muscles of the trunk, upper extremities and respiration. Although the paralyzed muscles are flaccid, there is little if any muscular atrophy; sensory disturbances are either slight or entirely absent; and the vesical and rectal sphincters are unaffected.

**Etiology.**—Very few diseases of the nervous system are involved in greater obscurity than is Landry's paralysis. While much has been surmised concerning its etiology and pathology, but little has been positively determined. It has been observed to attack males more frequently than females, and especially those between the ages of twenty and forty years. At the same time, no time of life is exempt, as examples of the disease have been observed in both childhood and old age. It has frequently been attributed to exposure to cold and damp; Watson, Cane, Service, and Batori reporting cases confirming this view of its etiology. Some authors claim that it may follow the acute infectious diseases, as typhus, typhoid fever, variola, and splenic fever; but it seems to me on reading their reports, that the affection described was really a multiple neuritis.

**Pathology and Morbid Anatomy.**—In the present state of our knowledge respecting this disease, its definition must include the proviso "without definitely determined anatomical changes." Numerous cases claimed to be Landry's paralysis have been reported in which neuritis of both the interstitial and parenchymatous varieties has been found. But these cannot be considered as examples of the disease under study. Then, too, a rapidly progressive neuritis cannot account for the symptoms of acute ascending paralysis; and cases have occurred and been carefully studied, and this but recently, in which absolutely no lesion was discovered by the most searching microscopic investigation. Notwithstanding this, Ross, from a study of ninety-three cases collected from literature, adheres to the neuritic origin of Landry's paralysis, and Nauwerck and Barth agree with him. Centanni found an interstitial neuritis in his case, and a peculiar bacillus in all the tissues. Carter has presented as a theory the possibility of an effusion into the central canal of the spinal cord, the effusion rising higher and higher, causes the peculiar progression of the symptoms.

Prevailing opinions of those most competent to judge, favor the idea that Landry's paralysis is due to some form of toxæmia, possibly arising

from a hitherto unknown micro-organism. Already it has been attributed to a bacillus affecting the sheaths of the peripheral nerves.

**Symptomatology.**—The disease is ushered in by premonitory symptoms consisting of certain vague sensory phenomena, as aching and soreness with tingling in the parts about to be paralyzed. Headache and backache are not unusual. Then the patient notices a weakness in both lower extremities, and this increases rapidly, even in a few hours, until the loss of power is absolute. The muscles first attacked are those of the feet; next in order come those of the legs; and finally those of the thighs. The lower extremities having been rendered functionless, the muscles of the trunk are next attacked, and rendered helpless; and then those of the arms are treated in like manner. The disease finally attacks the muscles of respiration, and thus carries off the patient.

According to Landry, the muscles are attacked in the following order: (1) Those moving the toes and feet; then the posterior muscles of the thigh and pelvis, and, lastly, the anterior and internal muscles of the thigh. (2) The muscles which move the fingers; then those which move the hands and the arm upon the scapula, and, lastly, the muscles which move the forearm on the arm. (3) The muscles of the trunk. (4) The muscles of respiration; then those of the tongue, pharynx and œsophagus. The disease does not always follow this typical course by any means. It may involve one side more severely than the other, or it may even begin in the upper extremities and spread downwards, thus making itself an acute descending paralysis.

The paralyzed muscles are flaccid, but do not show any but the slightest degree of muscular atrophy. The electrical reactions of the paralyzed muscles are normal, even in cases which have lasted some time and have progressed to convalescence.

The patellar tendon reflex is generally diminished, and often entirely destroyed, even in the earliest stage of the disease. In one case that recovered, reported by Weber (*Medical News*, Vol. XLI), it remained absent.

Sensory symptoms are always slight. They are limited usually to a slight soreness or discomfort in the early stages, with sense of formication.

The bladder and rectum are never affected.

**Diagnosis.**—The diseases with which acute ascending paralysis may be confounded are multiple neuritis, acute central myelitis, acute and subacute poliomyelitis anterior. In *multiple neuritis*, sensory symptoms are prominent. There are acute pains and sensitiveness to touch along the course of the inflamed nerves, and anæsthesia is marked. The reaction of degeneration is present, and the paralyzed muscles become atrophied.

*Acute central myelitis*, likewise, has prominent sensory disturbances.

The bladder and rectum are early affected. Bedsores, which are unknown in Landry's paralysis, are very common. Fever is a prominent symptom.

In *acute poliomyelitis anterior*, the paralysis reaches its height at once and makes no further progression. After a longer or shorter time, not infrequently within a few days, some of the affected muscles recover their functions in part. The paralyzed muscles atrophy at an early period. The reaction of degeneration is characteristic.

The temperature in Landry's paralysis is usually normal or slightly above that point. Marked fever is, therefore, a contraindication to its diagnosis.

Cases alleged to have arisen as a result of syphilis must not be regarded as examples of acute ascending paralysis. They pursue a different clinical course, responding readily to antisyphilitic medication.

**Prognosis.**—The course of this disease is almost uniformly unfavorable. It is very doubtful if those cases reported as cured are all really examples of acute ascending paralysis. Some of them have subsequently developed atrophic and spastic palsies. Certainly, where they followed closely on throat affections, and were attended by severe pains and anæsthesia, this doubt is a very healthy one. Usually a fatal result is reached in from three to twelve days. Cases that recover, last several weeks. Where the onward march of the disease is comparatively slow, the prognosis is more favorable than in cases progressing rapidly.

**Treatment.**—The therapeutics of acute ascending paralysis is largely conjectural. Little or nothing can be said of the results obtained thus far from treatment. One is obliged to follow theoretical indications. Absolute rest in bed from the very first is essential. At the same time, the diet must be easily assimilable and highly nourishing.

*Ledum*, *conium*, *aluminium*, *gelsemium*, *cocculus*, *rhus* and *secale* have been suggested as remedies from which to make a selection.



## LOCOMOTOR ATAXIA.

Locomotor ataxia is a disease of the spinal cord characterized by inco-ordination of gait, lightning pains, absence of the tendon reflexes, and other symptoms, and dependent upon a degeneration of the posterior root zones, often in association with a similar lesion in portions of the brain and peripheral nerves.

It is referred to in literature also as *tabes dorsalis*, posterior spinal sclerosis, progressive paralysis, and atrophy of the spinal cord.

**Etiology.**—Heredity exerts but little direct influence in the causation of ataxia. The patient may possibly have inherited a neurotic constitution, as shown by the existence of inebriety, neuralgia, mental disorders, etc., in other members of the family. Exceptional instances have been reported by Gowers, Dreschfeld, Carre and Trosseau, in which the disease has affected a number of persons belonging to the same family.

Sex evidently has an important bearing in locomotor ataxia. About 90 per cent. of the cases are males. One naturally attributes this great predisposition to the different modes of life led by men and women. Men are, of course, more exposed to cold, fatigue, and other deleterious influences.

Locomotor ataxia is essentially a disease of middle life. Most cases come under observation at from the thirty-fifth to the forty-fifth year. Careful clinical investigation often shows, however, that some of the earlier symptoms of the disorder had existed for ten or more years, and were generally attributed by the patient and his physician to some benign complaint.

Those who are exposed to great variations in temperature and moisture, as engineers, car conductors, car drivers, and travelling salesmen are especially liable to the disease. Soldiers, from lying on damp ground, making forced marches in cold weather, and because of their lives of anxiety and excitement, frequently become subjects of chronic spinal disorders, of which locomotor ataxia is the most frequent.

It is extremely doubtful if venereal excesses play as important a part in the production of ataxia as they have been credited with.

Of all the causes of ataxia, I believe that syphilis is by all odds the most important. So frequently do I obtain a syphilitic history in these cases, that I have almost come to consider the presence of such as a *sine qua non*, and when I fail to obtain the same, that the patient is either deceiving himself or his physician. The fact that women are subject to other syphilitic nervous diseases as are men, while they are rarely attacked

with ataxia, is a strong point against the above view of the relation between ataxia and syphilis. Those cases of ataxia with which I have met in women, have always been engrafted on a syphilitic constitution. The pathological nature of locomotor ataxia is essentially different from that usually observed in syphilitic nervous diseases. The views of authors differ widely on this subject, some holding that syphilis is the etiological factor in about 5 per cent. of the cases, while others, as Erb, hold that syphilis is at the bottom of the trouble in nine-tenths of the cases. And they even question the accuracy of the history in the remaining tenth, believing the possible pre-existence of a urethral chancre which escaped observation, or a mendacious patient. As in the case of other syphilitic nervous disorders, a history of very mild secondary symptoms is usually given.

For many years diphthëria, smallpox, and certain other acute infectious diseases, were credited with being causes of ataxia. Increased knowledge has taught us that these cases are in reality multiple neuritis, and are in no way related to the disorder under consideration.

**Symptoms.**—The sharp, fulgorant pains, or lightning pains as they are commonly called, are among the first symptoms of the disease to make their appearance. They are of a sharp, darting character, momentary in duration, and of great severity. They usually invade the lower part of the body first. When the disease is well advanced, there is hardly a part of the body which has not at one time or another been subject to their visits. They come with no regularity as regards time or distribution. On one occasion they appear at intervals of a few seconds or minutes, and after keeping the patient in torture for hours, days, or weeks, as the case may be, disappear, not to return again until perhaps after the lapse of a long period. Or the patient may be at comparative ease during the day, but after nightfall his sufferings are intense. Cold or damp weather, or a slight draught of air is frequently sufficient to re-excite these pains after their disappearance. In some cases these pains are confined to small well-defined areas of the skin, in the form of hyperæsthetic spots. In others, as shown by Charcot, they are associated with a herpetic eruption following the course of the nerve which supplies the area affected by the pains. The lightning pains are the most constant of all the symptoms of ataxia. It has been said that they tend to disappear when the ataxic stage has well developed. This is a mistake. The most severe suffering from them will often be observed in the terminal stages of the disease.

As constant a symptom as the lightning pains is the absence of the knee-jerk. This is also spoken of as Westphal's symptom. It is to be noted in the earliest stages of the disorder, and occurring in an individual who is known to have had a knee-jerk at one time, is decidedly suspicious. I would even make a more extreme statement, and look

with suspicion on all patients with syphilitic history and absent knee-jerks. If also lightning pains existed, I would then regard the nature of the case as indisputable. It is of course conceivable that there must be a stage at which the first evidence of the disease exists, and that evidence, so far as present knowledge goes, is found in the diminished or absent knee-jerk. It must be borne in mind that the knee-jerk is on rare occasions absent in the healthy; and likewise is a symptom of other diseases than ataxia, *e. g.*, diphtheritic paralysis, acute poliomyelitis, and peripheral nerve lesions.

The inco-ordination in gait, from which the disease derived its name, is a characteristic but not a constant feature. The patient exhibits certain irregularities in movement, and these are notably aggravated when the eyes are closed. If requested to stand with eyes closed and heels and toes approximated, his body sways to and fro, or if the disease be at all advanced, he staggers and falls. The position when attempting to maintain the erect posture is characteristic. All the extensor muscles are in a condition of tonic contraction. The contraction of the calf muscles causes an extension of the foot on the leg. The leg is rigidly extended on the thigh from contraction of the quadriceps. This attitude of the lower extremities produces a tendency on the part of the patient to fall backwards, which, however, is counteracted by what is apparently a voluntary contraction of the muscles which flex the trunk on the thigh. The body then is bent forward. Any tendency to fall in this direction is prevented by the support the patient derives from his cane. In this position the buttocks project backwards considerably, and a vertical line let fall from the great trochanters falls considerably behind the heel. In the early stages of the disease the only irregularity in gait noticeable is a certain amount of unsteadiness when rising from a recumbent or sitting posture, at first manifested at twilight, and later at all times. As the disease progresses, the patient keeps his eyes on his feet in order to aid his step. Owing to the strong contraction of the extensor muscles the patient often moves his limbs as one solid piece.

The movement of the limb in locomotion is then a forward and outward one. In beginning the step, the foot is brought into a position of dorsal flexion, and this position it maintains until it touches the floor again, which it does heel first, the sole of the foot following with a sort of thump. All the movements of locomotion are of a jerky character. Sometimes in making a step the patient lifts the foot much higher than necessary, not being able to judge distances accurately. As the disease progresses, walking and standing become more and more difficult until at last they are impossible. When the upper portion of the spinal cord is involved, the movements of the arms become impaired. At first the patient notices difficulty in reaching for or grasping an object, unless the gaze is fixed on it. Or he may observe that certain delicate movements,



which he could formerly perform with ease, become awkward. If he closes his eyes and attempts to touch a specified point of the face with the tip of one of his fingers, he fails in the effort. He experiences difficulty in buttoning his clothes. Finally, even such movements as those required during the act of feeding become impossible.

Throughout the course of the disease it will be found that the patient is able to exert an amount of muscular power altogether out of proportion to the helplessness exhibited.

Anæsthesia of the lower extremities is often observed. This asserts itself in the shape of a sensation as if the soles of the feet were padded, or as if the patient were walking on thick velvet carpet. The anæsthesia may involve all parts of the extremities and trunk alike, or it may exist in limited areas only, discoverable only after careful examination. It never exists to the high degree observed in cases of transverse myelitis.

Pain in the back, tenderness along the spine, girdle sensation about the waist, are rarely experienced in locomotor ataxia. When they do occur they indicate that there is more or less involvement of the spinal meninges.

Eye symptoms are prominent in this disease. Of these the Argyll-Robertson pupils, a condition in which the pupils respond normally during efforts at accommodation, but fail to react to the stimulus of light, are the most characteristic. This makes its appearance, as a rule, early in the disease. Myosis and mydriasis are likewise not uncommonly observed. Or again, there may be inequality of the pupils. Along with the failure of the light reaction of the pupils is a failure to respond to cutaneous stimulation of the temples.

Atrophy of the optic nerve is the most distressing eye-symptom of ataxia, in that it usually goes on to absolute blindness. Fortunately, in these cases, the disease does not progress to the ataxic stage for some unexplained, or unexplainable reason.

Paralysis of the ocular muscles is very frequently met with. In the majority of cases, it is just sufficient to produce diplopia without objective strabismus. Paralysis of the levator palpebræ superioris is also an occasional symptom. These ocular paralyses are apt to be but temporary. They may, however, appear and disappear a dozen times during the progress of the disease.

Mental symptoms do not belong to ataxia. Occasionally, they are superadded to the symptoms already mentioned, in which case it is more than probable general paralysis of the insane exists, and the spinal sclerosis is but symptomatic of a general degenerative process.

Bladder and rectal symptoms are by no means uncommon. Urinary incontinence is a frequent accompaniment of the late stages of the disease. In other cases, the patient is unable to empty his bladder completely by reason of deficient expulsive power. The residual urine is

apt, sooner or later, to set up cystitis. The rectal symptoms are not apt to be so prominent. They are usually manifested in the shape of deficient expulsive power.

Sexual desire in the early stage is unimpaired. It has even been said to be increased. In the late stages, all sexual power vanishes.

A variety of the lightning pains is found in the visceral crises, which complicate many cases of ataxia. The most frequently observed of these are the gastralgie attacks or gastric crises. The pains are paroxysmal, of a burning or lancinating character, described by the patient as if a red-hot fork had been plunged through the abdomen into the stomach. They are frequently associated with retching and vomiting. In some cases, the vomiting exists without the pain, and may be so severe that the entrance of the smallest quantity of water into the stomach is sufficient to renew it. These paroxysms of gastric pain usually last three or four days, and recur at intervals of a month or more. During their continuance, there may be marked irregularity of the heart's action. Their presence is usually attributed to irritation of the nuclei of the pneumogastric nerve by sclerosis of the adjacent nerve-tissues.

Renal crises sometimes occur. In these, paroxysms of pain appear in the lumbar region, and radiate to the epigastrium, and sometimes to the more deeply situated parts of the abdomen. The urine decreases in quantity, until almost complete suppression results. The pains frequently follow the course of the ureters. Similar pains often course along the urethra and rectum.

Pharyngeal symptoms are occasionally noted. They vary greatly in severity. They may consist only of a slight, spasmodic cough, with repeated respiratory efforts, the result of laryngeal irritation. At other times, attacks, similar to those of spasm of the glottis, come on. These paroxysms occasionally produce death. Usually, they last but a few moments, but they recur many times a day. Laryngoscopic examination usually gives negative results.

The joint lesions of ataxia are among the most remarkable of its phenomena. They usually appear in the early stages, though they are not of infrequent occurrence. When they do complicate the late stages, they generally attack the upper extremities, just at the time when the pathological process is about to invade that portion of the spinal cord. They are usually contemporary with the lightning pains. The knee-joint is the one most frequently affected, and then in decreasing order of frequency, the shoulder, elbow, hip, and wrist joints. The joint affection of ataxia consists of an hydrarthrosis associated with a diffused swelling of the surrounding tissues. It presents in its external aspects important points which distinguish it from ordinary synovitis. When, in the latter affection, the joints contain a large quantity of fluid, there are three points at which there is bulging of the joint capsule, namely, above the

patella and on each side of its ligament. This appearance in the joint would be present in ataxia but for the effusion in the periarticular tissues. We know that the effusion is submuscular because none of the ordinary signs of subcutaneous cedema are present, and the muscles respond normally to electric stimuli, which they would not do were they separated from the surface by the fluid. The tabetic arthropathy may disappear entirely in the course of a few months, leaving the function of the joint unimpaired, or it may go on to erosion of the cartilages and bone surfaces and result in complete destruction of the joint. There is a notable absence of pain. Buzzard has directed attention to the frequent association of arthropathy with gastric crisis.

The shafts of the bones, as well as the articular surfaces, may undergo pathological changes, so that they become brittle, and are liable to fracture from slight causes. Vallin, Puys and Lereboullet have observed in a few cases rarefaction of the alveolar border of the maxillary bones, as a result of which there is falling out of the teeth. Changes in the teeth themselves were met with by David. The anterior portions of the crowns were decayed, the altered parts becoming soft and of a reddish color.

Pitres has reported cases of ataxia in which there was a spontaneous falling off of the nails of the great toe unattended by pain or suppuration, and with no history of rheumatic cause preceding. The old nails were rapidly replaced by new ones.

Ball and Thiebierge, at the 1880 meeting of the International Medical Congress, referred to an occasional trophic symptom of ataxia, namely, perforating ulcer of the foot. These ulcers are liable to develop symmetrically. They usually appear early in the disease, and during the stage of the lightning pains. Frequently after the ulcer has healed it shows a tendency to develop when the patient resumes walking. Treves and some others have looked upon these ulcers as purely the result of mechanical pressure, and not as an essential trophic phenomenon.

**Diagnosis.**—The difficulty in the diagnosis of ataxia arises only in the recognition of the disorder in its early stages. The lightning pains are liable to be misinterpreted as rheumatic, because of their aggravation during damp or cold weather. They differ from rheumatic pains, however, in that they do not invade joints, in fact are not limited to any particular locality, are but momentary in duration, and are associated with absent knee-jerk.

The same character of pain is presented by many cases of general lithæmic neuralgia. These are apt to be sharp and darting, flying from place to place and aggravated by an impending change in the weather. Usually they are very superficial, and accompanied by hyperæsthesia.

From neuralgia, they are differentiated by the peculiar characters



above mentioned, and from the fact that the pains of neuralgia follow the course of definite nerve trunks. The absent knee-jerks are an important aid.

The early ocular symptoms, as diplopia, optic nerve atrophy, and pupillary changes, may be the initial evidence of the disease. In that case, they will be associated with absent knee-jerks, whereas if they are symptomatic of brain disease, or are strictly peripheral lesions, the knee-jerks will either be increased or normal.

Errors in diagnosis in which the joint lesions constitute apparently the only objective symptom are not infrequent. Such cases generally consult the surgeon, who looks upon the disease as a purely local one; and notwithstanding the anomalous character of the articular affection, more than one patient has been prepared for a resection of the joint ere a timely discovery of the true nature of the lesion was recognized.

The value of the ataxic gait with its well-known aggravation when closing the eyes, has been overestimated. True, it is present sooner or later in the vast majority of cases, but then there are so many cases in which it appears so late, that were the disease dependent upon it for recognition, the diagnosis could only be made when all hope of successful treatment must be abandoned. Its value is still further lessened in that it occurs in other diseases in which anæsthesia of the feet is a prominent condition.

Disseminated sclerosis of the spinal cord when the brunt of the disease is borne by the posterior columns, may present symptoms which render an accurate diagnosis well nigh impossible. The ataxia, the lightning pains, the optic atrophy, and the pupillary symptoms may all be present. Other symptoms indicative of affection of the antero-lateral columns and the anterior cornua may be manifested. When the sclerosed nodules appear in the brain, there will be tremor on voluntary movement, vertigo, headache, mental disturbance, and epileptiform attacks. As a rule the knee-jerk is present, if not exaggerated, and ankle clonus is obtainable.

Disease of the cerebellum may be confounded with locomotor ataxia, on account of the inco-ordination of gait sometimes present in the former. The gait, however, is more of a staggering nature, like that of a drunken man. The tendency is to fall backwards. The instability during progression is usually increased by vertigo, hence the patient is able to walk better with his eyes closed than with them open. The inco-ordination of movement is not present when the patient is lying down. There may also be pain in the occipital region with vomiting. The cutaneous sensibility, the knee-jerk, and the bladder and rectal reflexes are unimpaired. The lancinating pains of ataxia are absent. General convulsions may be present, as may also choked disk or optic neuritis.

Hysterical ataxia may be confounded with the organic form of the

disease. Here the course of the disease, together with the temperament of the patient, will assist in the diagnosis. The reflexes will be present or even exaggerated. Symptoms indicative of degeneration of peripheral nerves are absent. In hysterical ataxia the disordered gait is due to an impairment of the muscular sense, hence so long as the patient watches her movements her progress is good. In locomotor ataxia the disordered gait is only ameliorated by the aid of vision, and not entirely relieved as in the case of hysteria. Sight must not be lost of the fact that hysteria may co-exist with true locomotor ataxia.

Occasional difficulty will be met in differentiating certain pathological conditions spending their force on the vicinity of the postero-median columns, as tumor and meningitis. These cases present a much more rapid course than the characteristic type of ataxia. They generally give a syphilitic history and offer a more favorable prognosis.

**Prognosis.**—So far as curing in the sense of removing all symptoms is concerned, the prognosis of ataxia is unfavorable. Many cases may be improved, and many more so benefited that the disease ceases to progress. Few indeed are incapable of some relief from treatment. The natural course of the disease is a very slow one. The initial symptoms often exist for many years before the ataxic stage develops. Cases have been reported in which the symptoms had subsided, but the pathological lesions remained. Syphilitic cases taken early in their course offer a comparatively favorable prognosis. They can frequently be prevented from advancing to the ataxic stage.

**Treatment.**—The successful treatment of locomotor ataxia involves attention to details. Individual symptoms and the general disorder require attention. The lightning pains as a cause of most intense suffering demand prompt relief. Rest in bed is here a necessary measure. When the pains are not present, then a partial rest secured by undergoing as little exertion as possible, is important as a prophylactic agent. As palliatives when the pains have become severe, we find the coal-tar derivatives invaluable. Antipyrin is the most efficient of these; antifebrin or acetanilid and phenacetin coming next in order. Exalgin has also been highly praised, but I have never used it. Antipyrin in doses of three grains administered hypodermically will often give relief inside of three minutes. It must not be forgotten that this drug is highly irritating locally when thus administered. It is always wise in these cases to administer these drugs with care and discrimination at first; when the patient's tolerance is assured, then they may be given with more liberal hand. Under no circumstances should the initial dose be more than five grains; continued study of individual cases soon teaches the physician the proper dose and frequency of administration. Morphia must be the final result when other measures fail; but one must face the unpleasant fact that when once begun, its use will probably become

regular and the morphia habit started. I myself have never been obliged to use it since the introduction of the coal-tar derivatives. The application of chloroform or bisulphide of carbon to the painful areas has been recommended by different authorities.

Electricity is a necessary adjuvant in the treatment of ataxia. Both galvanism and faradism must be employed. Erb's combined galvanization of the spinal cord and sympathetic is the best central application. The negative electrode is placed over the superior cervical ganglion of the sympathetic on one side, while the positive is applied to the lumbar region of the spine to the opposite side of the spinous processes. A current of about from six to eight milliamperes is made to pass for one minute and a half. Then the electrodes are shifted to opposite sides. Thus, if the negative has been over the superior cervical ganglion of the left side, it is placed over the right; and the positive, which has been to the right of the spinous processes of the lumbar vertebræ, is changed over to the left. The current is allowed to pass through these parts for another minute and a half. Then the negative electrode is placed over the spinous processes of the cervical vertebræ, and the positive over those of the lumbar. This occupies another minute and a half. After this the limbs, and possibly the trunk, are thoroughly treated by the faradic brush, the current being as strong as can be conveniently borne. These sittings should be given on alternate days. Various symptoms arising in the course of the disease, as paralysis of certain groups of muscles, will call for electrical treatment which will differ in no manner from that employed in those cases where the same symptoms have arisen idiopathically or in connection with other pathological conditions.

*Argentum nitricum* is our standard remedy for locomotor ataxia, and should be administered with regularity in all cases in which clear indications for other remedies are not found. It produces all the cardinal symptoms of the disease, thus: Ataxic gait, with aggravation when the eyes are closed; atrophy of the optic nerve; contracted, dilated or unequal pupils; loss of pupillary reflexes; gastralgic attacks; retention of urine from paralysis of the bladder; complete loss of all sexual desire; priapism; shooting pains. It is interesting to note that this use of nitrate of silver is not confined to our school. Wunderlich long ago recommended it, and it has been a standard ataxic remedy among allopathic physicians ever since. Its homœopathic employment antedated Wunderlich by many years.

*Zincum* is likewise employed by both schools of practice. Its homœopathicity is unquestionable. A few years ago a series of cases of nervous disease attacking the workmen of the mines of Upper Silesia was reported by Schlochow. The disease only attacked those who had worked in the mines for a number of years or more. There were marked inco-ordination of gait and anæsthesia of the lower extremities. *Zincum* is especially



indicated when the numbness and formication of the lower extremities are pronounced; also when there are burning along the spine, pain at the last dorsal vertebra, and other symptoms indicative of spinal irritation. Sexual power is generally lost.

*Zincum phosphide* is used more by old-school physicians than by ourselves. The combination of phosphorus with zinc, certainly ought to make a very valuable spinal remedy.

*Alumina* has the ptosis and diplopia so frequently met with in the earlier stages of the affection; the patient is unable to walk in the dark without staggering; the soles of the feet feel as if padded; there is formication in the back and in the extremities; the nates go to sleep when sitting; the heels become numb with walking; and there is pain in the back as if a red-hot iron had been thrust into the spine. With aluminium metallicum Bœnninghausen cured four cases of locomotor ataxia which presented the above symptoms and also the following: Frequent dizziness; feeling in the face as if cobwebs were on it. The characteristic alumina constipation is also present.

*Phosphorus* is still another remedy employed by both schools of medicine. It is especially indicated in erethistic cases. Burning along the spine and in the affected extremities with formication is a prominent symptom. Extreme sexual excitement is present. When atrophy of the optic nerve is present, it is associated with flashes of light.

*Picric acid* is probably limited in its usefulness to cases in which inordinate sexual desire is present, and in the early stages. Marked asthenia is present.

*Belladonna* will be of value in some cases of lightning pain. It is indicated by reason of the suddenness of onset, and suddenness of disappearance of this symptom.

*Berberis* will be of value in the nephritic crises.

*Nitric acid* will relieve the lightning pains when they are of a sticking character, and in syphilitic constitutions.

*Iodide of potassium* will be of great help in the very early stages of syphilitic cases, when paralysis of single cranial nerves are present. It should then be given in large doses and persistently.

*Secale* has produced all the classical symptoms of locomotor ataxia as shown in the report of Tuzek concerning an epidemic of ergotism in Marburg a number of years ago. It is largely used by the old school because of its influence over the vascular system. It has not been much used by the homœopaths, notwithstanding its evident homœopathicity.

*Æsculus, causticum, colchicum, fluoric acid, gelsemium, kali bromatum, nux vomica, physostigma, rhus, stramonium, and sulphur* should also be studied.

## ATAXIC PARAPLEGIA.

**Definition.**—Ataxic paraplegia is a disease dependent upon a combined sclerosis of the posterior and lateral columns of the spinal cord, and characterized clinically by paraplegia associated with considerable inco-ordination of movement.

**Etiology.**—Its causes are essentially the same as those already given for spastic paraplegia. Its origin has been variously attributed to neurotic inheritance, exposure to cold, traumatism, overexertion, and sexual excesses. Syphilis is sometimes a cause, though with nothing like the frequency observed in locomotor ataxia. In many cases no cause whatever is traceable.

**Pathology and Morbid Anatomy.**—The morbid changes are found in the posterior and lateral columns. The lesion in the former situation, however, differs from that of ataxia in not involving the posterior root zones especially, and also in that it affects the dorsal cord greater in extent than or equally to the lumbar portions. In the lateral columns, it is principally the pyramidal tracts that are affected. Still it is not strictly systemic in character, for the neighboring tracts are also involved. The lateral limiting layer, and, less frequently, the direct cerebellar tract, may exhibit degenerative changes. Sometimes, the sclerosis takes an annular form, surrounding the periphery of the cord. The membranes and the gray matter of the cord are unimpaired. The morbid process exhibits in some cases a tendency to extend far beyond the tracts originally diseased, thus giving rise to a clinical picture not unlike that of chronic diffuse myelitis.

The paraplegia finds its explanation in the disease of the pyramidal tract, while the sclerosis of the posterior columns accounts for the ataxia. The excessive action of the reflexes is due to the involvement of the pyramidal tracts and the rarity with which the pathological process extends to the lumbar root zones.

**Symptomatology.**—In the majority of cases, the onset of ataxic paraplegia is very slow. There is first noticed a slight weakness or tired feeling on any exertion in the legs. This is accompanied by an unsteadiness or inco-ordination of gait. The patient finds himself unstable when standing with heels and toes approximated and eyes closed (static ataxia). The extra effort required in maintaining an equilibrium is especially manifest to the observer, when the patient stands in his bare feet, the tendons over the instep exhibiting the unsteadiness of the muscular contractions. All of these symptoms gradually increase in intensity

over a term of years, until complete paralysis finally develops. With the increased loss of power, the inco-ordination becomes a less prominent feature, and rigidity of the paralyzed limbs appears. Sensory symptoms are not prominent. The sharp lancinating pains of ataxia are present very exceptionally indeed. Pain in the back is of frequent occurrence, so much so as to deserve rating as a symptom of the disease. Occasionally the patient complains of dull pains in the legs, especially on fatigue. All the deep and superficial reflexes are greatly exaggerated; ankle-clonus is marked. The functions of the rectal and vesical sphincters and the sexual organs are often (though not necessarily) impaired to a marked degree. Optic nerve atrophy is rare; pupillary changes and ocular palsies are sometimes observed, but in syphilitic cases chiefly. Sometimes mental impairment is observed. This closely resembles that observed in general paralysis of the insane.

Individual cases will present more or less variations from the above type, according to the location in which the degenerative changes predominate; thus if the posterior columns are mostly diseased, ataxic symptoms are more obtrusive, and if the pyramidal tracts, the symptoms partake of more of the character of those of spastic paraplegia.

**Diagnosis.**—There seems to be danger of confounding this disease only with spastic paraplegia, locomotor ataxia, and tumor of the cerebellum. From *spastic paraplegia* it is distinguished by the inco-ordination of gait in the early stage; from *locomotor ataxia*, by the excessive tendon and superficial reflexes, and the absence of lightning pains; and from *cerebellar tumor* by the presence in most cases of the latter affection, of occipital headache, optic neuritis, and vomiting, and the absence of weakness of the legs.

**Prognosis.**—So far as cure is concerned, this is very unfavorable. The usual course of the disease is a very slow one. Exceptionally, cases which run a subacute course of from three to six months, are observed. Most of the patients die from the same causes that carry off the majority of cases of spinal cord degenerations, namely, cystitis and bedsores. With careful attention to hygienic and medicinal details these patients may live on for years, disabled it is true, but still fairly comfortable.

**Treatment.**—This is essentially the same as that outlined for locomotor ataxia. Many cases are greatly ameliorated by a prolonged course of electrical treatment.

**Hereditary Ataxic Paraplegia** has been described by Dr. Charles Henry Brown, of New York. It is a very rare condition, presenting the same general symptoms as above outlined, but occurring in children at about the age of puberty. The lesions preponderate in the lateral columns.



## SPASTIC PARAPLEGIA.

Spastic paraplegia is a form of paraplegia characterized by more or less rigidity of the paralyzed parts, and dependent upon degeneration of the pyramidal tracts in the spinal cord.

**Etiology.**—The majority of cases of spastic paraplegia occur in patients from twenty to forty years of age. Many of these are of a neuropathic constitution. Injuries and exposure to vicissitudes of weather are likewise believed to be occasional causes.

**Morbid Anatomy.**—Spastic paraplegia may be dependent upon either a primary or a secondary degeneration of the pyramidal tracts. As a primary disease, its existence has been denied, as of the many post-mortem examinations made on supposed cases, with few exceptions the lesion in the pyramidal tracts has been proven to be secondary to other changes. The objection has therefore been raised that more thorough examination, or improved technique, would probably have relegated these cases likewise to the class of secondary degenerations. If, however, we reason by analogy, there is every reason to acknowledge the existence of a primary spastic paraplegia; and, moreover, we observe cases clinically which can be readily separated from the known secondary cases.

**Symptoms.**—Speaking in a general way, the symptoms of spastic paraplegia consist of paralysis, associated with rigidity of the paralyzed parts and a most remarkable exaggeration of the deep and superficial reflexes. The disease is of slow onset always. There is first noticed a slight weakness of the lower extremities, which gradually progresses to disability. At the same time, the knee-jerks are found to be remarkably exaggerated, the slightest tap on the ligamentum patellæ being sufficient to produce a vigorous response. Rectus clonus and ankle clonus are obtainable. Rigidity appears, and is especially marked in the extensor muscles. A peculiarity of this rigidity is that it is greatest when the limb is extended. This characteristic explains a phenomenon which has been denominated by Buzzard and others the clasp-knife rigidity. Taking the rigid member in the hands and endeavoring to flex a joint, for example the knee, one finds that flexion to a certain degree is accomplished by the expenditure of quite considerable force. But having attained that point, force is no longer required to produce flexion. Conversely, when producing extension, up to a certain point, the aid of the hands is required, and then the limb seems to spring straight of itself.

The spastic gait is characteristic. The limbs are moved in one

piece. The toes scrape the ground. Sometimes, because of the strong adductor spasm, the limbs are drawn in front of each other, giving rise to the phenomenon called cross-legged progression. The tension put on the tendo-achilles, excites an ankle clonus, the tremulous movements of which are imparted to the gait.

Sensory disturbances are absent as a rule; if they do occur, they consist solely of sensations of numbness and formication. The sphincters are sometimes paralyzed. The electrical reactions of the paralyzed muscles are normal. The arms may be affected if the degeneration involves the cord above the cervical enlargement.

**Prognosis.**—This is very unfavorable. Gowers is inclined to take a more favorable view of these cases than are other authors, as he has seen some cases that have improved quite considerably. My own experience leads me to place these cases as the most unsatisfactory of all spinal scleroses.

**Diagnosis.**—There is more danger of confounding spastic paraplegia with hysterical paraplegia than with any other condition. In hysterical paralysis there is nearly always rigidity; but this rigidity is equally marked, whether the limb be wholly or partially flexed, or even extended.

The presence of marked sensory symptoms is always indicative of some other trouble than primary spastic paraplegia.

Ataxic paraplegia is differentiated from spastic paraplegia by the unsteady or inco-ordinate gait; amyotrophic lateral sclerosis by the muscular atrophy, and by the usual first appearance of the disease in the upper extremities.

**Treatment.**—Rest is a great element in the treatment of spastic paraplegia. This should be as absolute as the patient's circumstances will permit. Massage is an important measure. The muscles should be made tense and then rubbed upwards. Electricity should not be employed, as galvanism is useless, and peripheral faradization harmful.

As to remedies, *lathyrus*, *argentum nitricum*, *phosphorus*, *zincum*, *nux vomica* and *strychnia* should be studied.

## FRIEDREICH'S ATAXIA.

**Synonyms.**—Friedreich's disease; hereditary ataxia; hereditary ataxic paraplegia.

**Definition.**—Friedreich's ataxia is a form of ataxia resembling in some few of its features tabes dorsalis, but especially characterized by its remarkable tendency to attack several children of the same family. The inco-ordination commencing in the lower extremities, extends to the arms, and this in turn is followed by disturbances of speech and nystagmus.

**Etiology.**—While it is customary to speak of Friedreich's disease as hereditary ataxia, strictly speaking, this is a mistake, for it should be regarded simply as a family disease. The first manifestations of the disorder appear very early in life, often, indeed, before the child has begun to walk. In some very exceptional instances its advent has been postponed until the twenty-fifth year. It has been said, with apparently good reason, that all cases occurring in the same family make their beginning within two or three years of the same age. The disease occurs with equal frequency in the two sexes. In some families mostly males, and in others mostly females are affected. It has been claimed that the only cause of Friedreich's ataxia is the hereditary predisposition. Still cases have been observed in which an exciting cause has apparently been at work. Thus the disease has developed after typhoid fever, scarlatina, whooping cough, and other infectious diseases, and in persons of tubercular diathesis.

**Pathology and Morbid Anatomy.**—The anatomical changes in Friedreich's ataxia consist of a sclerosis of the posterior and lateral columns of the cord. Mendel's observations of a single case show that the peripheral nerves and cerebellum may also be affected. As to the nature of the sclerosis, opinions differ. Until within a very recent period it was held that this sclerosis was of the connective tissue type. Dejerine and Letulle have apparently shown, however, that the lesion of the posterior columns is of the neuroglial type of sclerosis, while that of the lateral columns is of the vascular or connective tissue variety, and secondary to the former. Auscher's examination of the peripheral nerves revealed a large number of fibres which resembled embryonic nerve fibres.

**Symptomatology.**—The onset of the disease is always gradual. It sometimes happens that its first manifestations, which are always referred to the lower extremities, come on before the patient has learned to walk.



The trouble is then very apt to be overlooked, and the case regarded as one of retarded development. In those who have already learned to walk, the onset of the disease is characterized by an inco-ordination of gait, which resembles more the gait of intoxication than that of locomotor ataxia. It may or may not be aggravated when the eyes are closed. Next, the arms are affected, and usually within a short time, as compared with the progress noted in other degenerative spinal affections. Voluntary movements of the arms become irregular and jerky. Nearly always the knee-jerks are early destroyed. Very exceptionally, and then only in atypical cases, they are exaggerated. The further progress of the disease is characterized by involvement of the movements of the head and neck. These become irregular and jerky, and there may even be an irregular tremor. Speech becomes affected; syllables are omitted and utterance is slow and hesitating. Nystagmus, observed especially when the patient is looking to one side, is nearly always present. Optic nerve atrophy and pupillary symptoms are absent.

Sensory symptoms do not present themselves with any regularity. They consist of dull pains, slight anæsthesia, delay in sensory conduction and hyperalgesia.

Various deformities mark the later stages of hereditary ataxia. Lateral curvature of the spine is commonly present, and may or may not be associated with angular deformity. Talipes equinus or equinovarus sometimes appears. A characteristic deformity of the feet is the dorsal flexion of the great toe on its phalanx.

**Diagnosis.**—*Disseminated sclerosis* is to be differentiated from hereditary ataxia by its more rapid course, the wider range of the disorderly movements and the peculiarities of the speech. *Disseminated sclerosis* is accompanied by the "scanning speech," in which syllables are unduly separated.

From *cerebellar disease* Friedreich's ataxia may be distinguished by the presence of head symptoms in the former, and involvement of the arms in the latter.

**Prognosis.**—Friedreich's ataxia is incurable. It always runs a very protracted course, the majority of cases dying of some intercurrent disorder. The usual duration of the disease is from ten to thirty years.

**Treatment.**—This is, of course, highly unsatisfactory. It may be conducted on the general principles suggested in the pages devoted to the treatment of locomotor ataxia. Ladame thinks he has seen considerable benefit follow the application of galvanism to the spine three times weekly. The electrodes should be large ones, and the strength of the current from ten to fourteen milliamperes. The treatment must be kept up uninterruptedly, except for short intermissions, for several years.

## PROGRESSIVE BULBAR PARALYSIS.

**Definition.**—Progressive bulbar paralysis, or progressive glosso-labio-laryngeal paralysis, as it is also called, is, as its name indicates, a paralysis involving the muscles of the tongue, lips, pharynx and larynx, and dependent upon a degeneration of the motor nuclei of the medulla oblongata.

Two varieties of bulbar paralysis are recognized, the acute and the chronic. The latter only receives the name which heads this article. In the acute form, the symptoms are dependent upon hæmorrhage, inflammation, thrombosis, or embolism affecting the medulla or adjacent parts. Usually in such cases certain premonitory symptoms, as vertigo, headache, pain in the nape of the neck, and vomiting, are noted. Paralysis of the various muscles receiving their nerve supply from the bulb quickly follows. The distribution and extent of these paralyses differ in different cases according to the situation and severity of the lesion. In complete cases, the tongue, lips, pharynx and larynx are paralyzed bilaterally, rendering speech and deglutition absolutely impossible. Breathing even may be affected, becoming rapid and irregular. Death generally takes place within from three to ten days. Some few cases do not end fatally. Three such occurring in my experience arose from syphilitic disease, and I believe were dependent upon a syphilitic basilar meningitis. In each case the disease after the subsidence of the general symptoms accompanying the rapid onset pursued the ordinary progressive course of the chronic disorder.

**Etiology.**—Progressive bulbar paralysis rarely occurs before the age of forty; and is fairly frequent from that age up to the seventieth year. Males are more frequently affected than females, probably because they are more frequently subjected to the exciting causes of the disease, as exposure to wet and cold, exertion, worry, etc. Syphilis has been assigned as the cause of some cases; but careful investigation of these will oftentimes show that they present clinical features which clearly distinguish them from the primarily degenerative type of the disease.

**Symptoms.**—The onset of progressive bulbar paralysis is very insidious, it often being ushered in by such indefinite subjective symptoms as discomfort or feeling of pressure in the nape of the neck, slight vertigo, general languor, and other symptoms of like doubtful significance. The paralysis of the tongue is first manifested by a certain degree of difficulty in talking, or possibly by local fatigue after speaking for a long time without resting. According to the special muscles

first attacked, will the difficulty be noticed in attempting to pronounce this or that class of consonant or vowel sounds. Sometimes the lips are the first affected, and sometimes the larynx. Usually, however, the tongue is the first to suffer. Finally dysphagia appears. Then there may be especial difficulty in swallowing solids or liquids or both according to circumstances. If the tongue is seriously disabled, then the patient will find it difficult to properly prepare the bolus of food, or even push it back into the pharynx without the aid of his hands. If, on the other hand, the muscles which close the glottis are affected, then attempts at swallowing liquids will be accompanied by choking and strangling. It may happen that the act of swallowing liquids will be followed by regurgitation through the nose, owing to paralysis of the soft palate. When the laryngeal muscles are severely involved, the voice becomes nearly lost. It is rare for the paralysis of the laryngeal muscles to proceed to the extreme extent observed in the case of the muscles of the tongue. The paralysis of the orbicularis oris makes it impossible for the patient to pucker the lips, or to make such motions as those involved in blowing, whistling, kissing, etc. It is also impossible for the patient to retain saliva in the mouth; thus there is a more or less constant dribbling, necessitating the retention of the handkerchief to the mouth as a matter of cleanliness. The quantity of saliva is believed to be increased in these cases, and its composition altered so as to make it more viscous than normal.

The paralyzed muscles undergo a high degree of atrophy. The lips become thin; the tongue wastes, and creases and folds are noticed on its surface. The local reflex excitability of the parts is more or less destroyed, although common sensibility is retained to perfection. The affected muscles exhibit the reaction of degeneration; but the nerves going to those muscles present normal electrical reactions. Intelligence is preserved to the end. The pneumogastric nerve is sometimes involved in the terminal stages, as is exhibited by certain cardiac symptoms, rapid pulse, syncope, etc. These phenomena may appear unexpectedly on the patient unduly exerting himself, as in rising from bed.

The terminal stage of bulbar paralysis is not infrequently complicated by general muscular atrophy or by amyotrophic lateral sclerosis. These affections are closely allied to it. It may even be that bulbar paralysis complicates the terminal stage of these varieties of muscular atrophy.

**The Prognosis** of progressive bulbar paralysis is absolutely unfavorable. No reliable report of cure of a case has ever been made. Some few cases have been stopped in their onward progress, or made to proceed less slowly.

**Treatment.**—Faradization of the muscles of the palate and tongue and the use of strong galvanic currents through the neck and spine have



greatly benefited two cases under the care of Kussmaul. Where dysphagia is a prominent symptom, electrical applications may be made so as to promote deglutition. The positive pole should be fixed to the nape of the neck, while the negative is rapidly moved downward over the side of the larynx. When swallowing becomes impossible, then recourse must be had to the stomach tube for feeding purposes. Food then must be in a more or less liquid form. The frequent use of the tube may injure the parts, so as a matter of precaution rectal alimentation may also be employed.

*Anacardium* has been recommended, but on what grounds it is difficult to understand. *Plumbum*, owing to its special action on nuclear cells, should be as distinctively homœopathic to this affection as it is to progressive muscular atrophy. *Causticum* from its utility in paralysis of single parts or single nerves has been recommended by Farrington in this disease, although he expresses an earnest doubt that it will accomplish anything. It is a suggestion merely, a straw to grasp in a desperate situation. *Argentum nitricum*, *phosphorus* and *mercury*, may also be studied.

## AMYOTROPHIC LATERAL SCLEROSIS.

**Synonyms.**—Sclerose laterale amyotrophique; spastische Spinal-paralyse mit Muskelatrophie.

**Definition.**—Amyotrophic lateral sclerosis is a disease of the spinal cord characterized clinically by a gradually progressive paralysis associated with muscular atrophy and rigidity; and pathologically by degeneration of the pyramidal tracts and the anterior cornua of the spinal cord.

**Etiology.**—The etiology of this disease may be stated to be the same as that of spinal muscular atrophy. Krafft-Ebing reports one case in a woman attributed to repeated falls on the back.

**Pathology and Morbid Anatomy.**—The pathology of amyotrophic sclerosis is the subject of considerable difference of opinion. The commonly accepted view is that held by Charcot, Gray, Oppenheimer, Destree, and Le Bœuf, to the effect that the disease begins in a degeneration of the lateral and anterior pyramidal tracts and ganglion cells in the anterior cornua. Oppenheimer's observations show that the degeneration of the pyramidal tracts is primary, and that of the anterior cornua occurs later on. Some have claimed that the pyramidal tract degeneration is secondary to a lesion above, but inasmuch as many cases are observed in which the brain is perfectly normal, this view has not been accepted, however strong may be the reasons which urge it. In many cases degeneration of the nerve nuclei in the medulla are found. Gowers differs from the above-named authorities and considers amyotrophic sclerosis but a variety of progressive muscular atrophy. Marie and Mingazzini found in their cases, that the degeneration was not limited to the pyramidal tracts, but that it was disseminated throughout the entire transverse section of the cord. Destree and Le Bœuf in their cases found degeneration of the remaining portions of the antero-lateral fasciculi and lesions of the columns of Goll.

**Symptomatology.**—With the lesions in the situations above mentioned the symptomatology of this affection can be readily surmised. According as the pyramidal tracts or the anterior horns are relatively more or less affected, so will spastic paralysis or muscular atrophy predominate. Usually the first symptoms of the disease manifest themselves in the upper extremities, sometimes in the fingers, in the shape of sensations of formication and numbness. This is shortly followed by motor weakness associated with a more or less rapidly progressive muscular wasting. The disability generally affects all the muscles of the affected

limbs alike; the atrophy involves the muscles as a whole, and not fibre by fibre, as in the regular types of muscular atrophy. Fibrillary twitchings are nearly always observed in the affected muscles; this is denied by some writers. Soon muscular rigidity is noted, and this increases in intensity until it may amount to absolute contracture. When the spastic stage is fully developed, the arms become firmly drawn to the body, the forearms bent on the arm, the hands pronated, and the fingers strongly flexed.

In the second stage of the disease the legs become involved. Here paraplegia with rigidity is the early prominent symptom. Atrophy of the leg muscles does not usually occur until comparatively late.

In the third stage, a paralysis of the muscles of the lips, tongue, pharynx and larynx occurs by reason of extension of the degeneration to the medulla. In the terminal stage, the mind becomes affected; memory fails; the patient becomes childish and credulity is a prominent characteristic; emotional excitability is noted. Finally even complete dementia may result.

Throughout the disease the deep reflexes are increased. The knee-jerks are exaggerated and the tendon reflexes are readily obtainable at the elbows and wrists. Ankle clonus is present. In some cases the spastic phenomena are absent, in which case the same increase in the tendon reactions is still observed. When the atrophy advances to an extreme degree, the spastic condition diminishes in a great measure. If, however, the latter has existed very long, the permanent alterations in the structure of the muscle resulting therefrom, makes the deformities permanent.

**Diagnosis.**—Amyotrophic lateral sclerosis may be confounded with progressive muscular atrophy, poliomyelitis anterior and myelitis. From *progressive muscular atrophy* it is differentiated by the contracture of the paralyzed muscles, and the marked exaggeration of the deep reflexes. The paralysis is the primary symptom, and the atrophy, secondary. In progressive muscular atrophy the loss of power is in direct proportion to the degree of wasting.

*Acute poliomyelitis anterior* presents a clinical course entirely different from that of amyotrophic lateral sclerosis. The paralysis is of rapid onset, the muscular atrophy comes on two or three weeks subsequently, the deep and superficial reflexes are destroyed, and the contractures are late, and the result of the unantagonized action of muscles still in possession of part of their functions.

*Myelitis* has marked sensory symptoms, and the bladder and rectum are early affected.

**Prognosis.**—The prognosis of amyotrophic lateral sclerosis is decidedly unfavorable. It almost invariably proceeds to a fatal termination in from one to three years. Some cases lasting ten years have been reported.



**Treatment.**—Treatment seems to avail little. The most efficient agent is massage. Other measures can only be adopted on general principles, as suggested by symptoms as they arise. Remedies acting upon the anterior cornua and the pyramidal tracts are the ones which should be thought of. They are *arsenicum*, *strychnia*, *plumbum*, *lathyrus*, *picric acid*, *argentum nitricum*, and *sulphur*.

## CAISSON DISEASE.

The caisson disease is an affection produced by a too sudden return to the surface after prolonged working under great atmospheric pressure, as occurs in caissons and diving bells. It is characterized by pains in different portions of the body, nausea and vomiting, and paralysis of motion and sensation, generally in the lower extremities.

**Etiology.**—The essential point in the causation of caisson disease is the sudden withdrawal of the patient from great to ordinary atmospheric pressure, especially in those who are not accustomed to such changes. The greater the air pressure in the caisson, and the more rapid the transfer to the outside world, the more likely is the disease to ensue, and the more severe are the symptoms apt to be. It very rarely occurs where the workmen are exposed to but one atmosphere or less. Personal predisposition and carelessness have much to do with the production of the trouble, for it has been observed with especial frequency in persons of corpulent build, in those addicted to alcoholic indulgence and excesses generally, and in the subjects of chronic heart and renal disease. Elderly men seem to be more liable than are the young ones. Old hands possess a certain degree of immunity; still this is only relative, for under favoring circumstances, they also may be attacked.

**Pathology and Morbid Anatomy.**—The prolonged subjection of the workmen to air pressure ranging from fifteen to fifty pounds to the square inch, results in the blood leaving the skin and superficial structures of the body, and accumulating in the internal organs. On return to ordinary atmospheric surroundings, this pressure is abruptly relieved, and the blood as suddenly tends to return to the surface. Congestions and small hæmorrhages follow; and inasmuch as the spinal cord and brain are enclosed in bony cases, and are thereby surrounded by inelastic coverings, they are the least fitted to withstand the bad influences of the changed conditions. They therefore bear the brunt of the disorder. Bert has offered another theory of the causation of caisson disease. He claims that the increased atmospheric pressure leads to the excessive absorption of nitrogen, which is liberated on the cessation of the pressure, appearing in the vessels of the cord as bubbles, and so obstructing those of fine calibre.

Autopsies disclose the changes incidental to disseminated myelitis. The lesions are found irregularly distributed through the affected segments of the cord, but mainly in the lumbar enlargement. Congestions and hæmorrhagic extravasations are also observed.

**Symptomatology.**—The first manifestations of the disease are observed in half an hour or thereabouts after coming to the surface. The skin becomes cool, the patient breaks out into a sweat, the pulse becomes small and compressible, and pains appear. This latter symptom is especially marked in the lower extremities, although, according to Corning, pains may be found also in the elbows, deltoid muscle, the lumbar region of the spine, and the ears. They may be either sudden or gradual in onset, paroxysmal or steady. Some cases exhibit epigastric pain associated with nausea and vomiting, as their prominent feature. Vomiting may be the sole symptom, and is then usually of cerebral origin. Many cases suffer only from these sensory symptoms, and then begin to improve. Others, less fortunate, go on to the paralytic stage. This is marked by loss of motor and sensory function in the lower extremities, sometimes consisting of a mere weakness, and in severe cases, of complete paraplegia. The pains do not ameliorate with the advent of anaesthesia. Paralysis of the sphincters is a frequent symptom, even in cases in which the paraplegia is of mild degree. When the functions of the rectum and bladder are restored, fæces and urine are often passed unconsciously, owing to the local anaesthesia.

Sometimes, the disease assumes a cerebral type. The paralysis is then hemiplegic in distribution, and is accompanied by mental impairment, as shown by coma, incoherence of speech, and syncope, and by such cerebral symptoms as vertigo and diplopia.

**Prognosis.**—The great majority of cases make excellent recoveries in periods ranging from a few hours to six weeks. Especially in cases in which pain constitutes the main symptom, does rapid return to health occur. Even the paralysis may disappear within twelve hours. Some cases develop all the dangerous symptoms of myelitis and die in a short time, or recover only after an illness of many weeks. The outlook is especially unfavorable in old, corpulent, and dissipated subjects.

**Treatment.**—The treatment of the caisson disease should be a preventive one. Those unused to caisson work should gradually accustom themselves to the increased atmospheric pressure, and observe care not to remain exposed for too long a time at first. They should, moreover, see to it, that their return to normal atmospheric conditions is made by degrees. In “coming out” it is believed to be safe practice to allow five minutes in each lock for each atmosphere of pressure.

The severe suffering even in cases of short duration, calls for palliation at times; morphia hypodermically being the most available analgesic. Pains in the extremities may be relieved by immersion of the parts in hot water. Jaminer found the administration of alcohol with ginger of great use for the epigastric pains. It has been found that the partaking of food before entering the caisson is a very important preventive of accident. On coming to the surface, workmen should invari-



ably rest for a short time; *above all things, they should not think of making any undue exertion.* Return to the caisson has been recommended as a preventive of further trouble, on the advent of the initial symptoms. Severe cases call for the same treatment as recommended for myelitis. As to remedies, *arnica*, *belladonna*, *bryonia*, *rhus*, *nux vomica*, and *causticum*, are the most frequently indicated.

## SYRINGOMYELIA.

**Definition.**—Syringomyelia is a disease of the spinal cord believed to be characterized clinically by the association of peculiar sensory and trophic phenomena, and dependent pathologically upon the formation of cavities in the spinal cord, which cavities occur by reason of embryonal defects, and have walls composed of gliomatous tissue.

Hirt makes a distinction between cavities formed within the substance of the cord itself, and dilatations of the central canal, the latter being designated by him, *hydrorrhachis*.

**Etiology.**—Syringomyelia is at present regarded as a very rare disease, so rare indeed, as to be a clinical curiosity. Its etiology is not well understood. As matters of fact, it has been observed that more cases occur in men than in women, and that the symptoms of the disease become obtrusive between the ages of fourteen or fifteen and twenty-five. It has also been observed that the majority of cases occur among the laboring classes. Syringomyelia has also been known to follow pregnancy, traumatism, and the acute infectious diseases.

**Pathology and Morbid Anatomy.**—The central lesion in syringomyelia is the formation of one or more cavities within the substance of the spinal cord, generally within the horns of gray matter. These cavities vary greatly in size and shape in individual cases. Sometimes they are such as to convert the cord into a thin-walled tube. The contained fluid may be either liquid or gelatinous.

The primary lesion, that is the one to which the cavity formation is due, is a neoplastic hyperplasia of the neuroglia of the gray matter; secondary changes are set up within this substance, and cavities result.

Peripherally, are observed atrophic changes in muscles, bones, skin, joints, and peripheral nerves.

**Symptomatology.**—While a clinical picture of syringomyelia has been constructed, and has received the sanction of authority, it must be remembered that many cases do not coincide with this symptomatic totality. Autopsies have revealed the presence of cavities within the spinal cord, when the symptoms during life were not at all suggestive of the existence of such a condition; and, on the other hand, cases have occurred, in which the peculiar sensory and trophic phenomena were present, but in which the diagnosis of syringomyelia failed of confirmation at the autopsy.

The onset of the disease is characterized by its insidiousness, the first manifestations being observed in the upper extremities. There

appear pains in the back of the neck, and these are accompanied by paræsthesiæ, numbness, and wasting in the arms. Thus far, the symptoms bear a close resemblance to those of amyotrophic lateral sclerosis. The sensory disturbances, on closer examination, are found to present distinguishing features, for while sensibility to pain and temperature are diminished, or even destroyed, that to touch is preserved. Finally, the legs become affected with spastic paraplegia, but without atrophy or sensory disturbance.

The sensory disturbances of syringomyelia are not always dissociated in the manner above described. Sometimes, thermic or muscular sensibility is preserved, or even hyperæsthetic. Sometimes, there is perversion of these senses.

The motor symptoms in some cases, instead of partaking of the nature of paralysis, assume the characteristics of ataxia or inco-ordination. The paralysis in the upper extremities is rapidly followed by muscular atrophy and remarkable deformities. The muscles of the back participate early in the paralysis and atrophy, and thus there ensues marked curvature of the spine, especially in the dorso-lumbar region, with the convexity of the curvature to the left.

The trophic changes, observed in the skin, consist of glossiness of the skin, bullous, eczematous, or herpetic eruptions, and even of perforating ulcers and gangrene.

The bones also exhibit trophic changes, as exhibited by the arthropathy, and the tendency to fracture from slight causes.

The late stages of the disease may be characterized by evidences of extension of the lesion to the medulla, and by paralysis of the rectum and bladder.

**Diagnosis.**—Syringomyelia may be confounded with cervical pachymeningitis, from which it differs by being much less painful, and the peculiar dissociation of the sensory disturbances. Age is an important clinical factor in diagnosis, as the majority of cases of syringomyelia occur in adolescents or young adults. The main symptoms on which reliance should be placed, are the muscular atrophy, the dissociation of sensory disturbances, and the spinal curvatures.

The relation between leprosy and syringomyelia has been much discussed of late years, many prominent men assuming that the two diseases are identical. While the resemblances between anæsthetic leprosy and syringomyelia are indeed remarkable, it seems to me that the identity of the two conditions has been far from proven.

**Prognosis.**—The prognosis of syringomyelia is bad.

**Treatment.**—Treatment cannot be directed to the pathological condition in these cases with the slightest chance of success. One must combat symptoms as they arise. In the management of the patient, it must be remembered that these cases, not feeling the effects of heat, may readily burn themselves severely.



## MORVAN'S DISEASE.

This is believed to be a variety of syringomyelia, complicated by a peripheral neuritis. The probabilities are that there are two varieties of Morvan's disease, one with and the other without syringomyelia. Both varieties are characterized symptomatically by painless whitlows on the fingers, involving first one hand and then the other, accompanied by brittleness of the nails, which may drop off. Joint complications may ensue. This type of syringomyelia has been observed only among the fishermen of Brittany.

The remedies suggested by Lilienthal as likely to prove useful both in this condition and syringomyelia are *thuja*, *silicea*, *natrum sulph.*, *meze-reum*, *selenium*, *graphites* and *sepia*.

# FUNCTIONAL AFFECTIONS OF THE NERVOUS SYSTEM.

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## HYSTERIA.

To give a comprehensive and accurate definition of the term hysteria is not an easy matter. Authors in attempting it have either gone into profuseness of detail or have lacked accuracy, no one definition suiting all cases. Perhaps the best one thus far offered is that of Möbius, adopted also by Osler in his text-book, namely: A state in which ideas control the body and produce morbid changes in its functions.

**Etiology.**—The first thing to impress the observer respecting the etiology of hysteria is the remarkable predominance of the disease in the female sex, a predominance so great that at one time it was a current belief that hysteria was exclusively a disease of women. Hysteria is now recognized as affecting males also, probably 10 per cent. of the cases occurring in the latter. The frequency with which hysteria occurs among females should not lead to too broad a generalization and establish in the mind of the practitioner that it is dependent upon lesions of the female genital organs. On the contrary it should be recognized as of frequent occurrence in women because of the sensitive nervous organizations possessed by them. On this subject more will be said very shortly.

Racial predisposition seems to be as fully marked as that of sex. Hysteria is practically unknown among barbarous peoples; it is a product of the development of the emotional nature incident to civilization. This statement must not be interpreted to mean that hysteria is of most frequent occurrence in the most enlightened people, but rather that it occurs with especial frequency among civilized nations in whom the emotional nature is developed at the expense of the mental and physical, *e. g.*, the French and the nations of Southern Europe. In England and Northern Europe, while very commonly observed, the disease does not assume the severe type observed in the countries first named. It is quite common in America, its main manifestations here closely resembling those observed in England.

The majority of cases are observed in young adults. Over four-fifths of the cases exhibit the first evidences of the disease prior to the age of thirty. Puberty is a favorite time for the first manifestations. No age, however, is exempt. Cases have been observed in young infants and in those well advanced in years.

The most important cause of hysteria is a neuropathic inheritance. In very many instances, a family history of hysteria, insanity, epilepsy, hypochondriasis, and other nervous disorders, is obtainable. Of equally great importance is the injudicious training to which many children and young people are subjected. Taught by force of example to watch and magnify every unimportant ache and ail, and to direct the mind to self, permitted to live a life of selfishness without the pursuit of any praiseworthy aim or occupation, very slight emotional influences are sufficient to call forth the wildest hysterical manifestations. As to these emotional influences, they may consist of real or fancied disappointments,—altogether too often the latter only. An unhappy love affair, a mesalliance in marriage, the ordinary vexations of home life, loss of money and friends, or a single false step, is sufficient in those predisposed to light the disease into activity. One does not always succeed in discovering the existence of these causes even when present, unless he has fully gained the confidence of his patient. To win such confidence, one must possess himself of experience and tact in the management of hysterical patients. Then will he be convinced of the importance of moral and emotional causes in the production of this protean disease. No case has been thoroughly examined unless these causes have been searched for. With increasing experience one is led more and more to them as the origin of the ailment, and less and less to hypothetical reflex sources such as the uterus and “the orifices.”

Akin to emotional causes are depressing agencies. Thus excessive devotion to work is not an infrequent cause of hysteria in those of neuropathic constitutions. Especially is this true when the overwork has been combined with worry and anxiety. Asthenia following acute illnesses, *e. g.*, typhoid fever, or resulting from insufficient or improper feeding, is in many instances the sole cause.

There is no definite relation between the state of the general health and the generation of hysteria. The disease occurs many times in those apparently strong, and it attacks the anæmic and cachectic. Naturally hysterical symptoms appear in the debilitated, when the predisposition to the same exists. Those inclined to hysteria exhibit certain psychical peculiarities. For the most part, they are people of lively disposition, subject to powerful and changing emotions, and possessed of but little force of will. Aside from these mental abnormalities, they are often persons of considerable natural ability, and are nearly always possessed of a most intense egotism.

We now come to the discussion of a question in the etiology of hysteria concerning which very strong views have been expressed on either side, and that is the relation of hysteria to utero-ovarian disease. In former years hysteria was regarded by all as a disease of the female generative system, an opinion that gave it the name by which it is gen-



erally known. Recent clinical observations show most conclusively the fallacy of such notions, and assign the cause of hysteria to the nervous system. In favor of the utero-ovarian origin of hysteria are the following statements: In many instances, an alleged ovarian tenderness is present, aggravation of which by pressure is sufficient to bring on a paroxysm; during paroxysms ovarian pressure sometimes puts an end to the seizure; the existence of utero-ovarian disease in a very large percentage of the cases; the alleged cure of the hysteria by oöphorectomy or other surgical operations on the genital organs; the cure of hysteria by the use of pessaries and other local therapeutic measures. The so-called ovarian pain is by no means a proof of ovarian disease. Commonly present in hysterical cases, it exists even in those in whom the ovaries have been removed, and has been observed in the usual position when bimanual examination demonstrates the corresponding ovary to be elsewhere. In very many cases it is not pressure on the ovaries alone that is sufficient to precipitate or put an end to an attack, but pressure in quite widely separated localities has a like effect. The improvement following oöphorectomy and other surgical operations is not proof of the special value of those procedures. It must be remembered that this operation is one involving a profound moral effect. Owing to the magnitude and danger of the operation, the patient is secluded from her friends. The latter, moreover, entertaining a profound respect for the danger of a major surgical operation, obey the surgeon's injunctions to the letter; the patient is kept at absolute rest for a prolonged period, and for some time following the operation is subjected to certain rules of living. Her mind is taken from her nervous symptoms and made to dwell on an actual physical condition, and so improvement takes place. No less injurious are the minor gynæcological procedures. Directing as they do the patient's mind to a portion of her body which is in the main a mystery to her, they afford a source of more or less constant worry, and not infrequently provoke hysteria, when a little rest or sound advice might have acted as a preventive. So strongly rooted has become popular prejudice in favor of disorders of the genital organs as a cause of nervous affections, that much harm is being daily, I might almost say hourly, done by gynæcologists, *i. e.*, by men whose main acquaintance with the domain of medicine is confined to a partial knowledge of the pelvic viscera of women. I might also speak in stronger terms, and say it is my firm belief, founded on not a small experience, that more harm has been done by this unfortunate gynæcological superstition than there has come good from the wonderful advances in abdominal surgery. I have seen epileptics robbed of their ovaries on the slightest pretext; I have seen others with the same disorder promised a cure on the repair of a lacerated perineum or dilatation of the cervix; I have seen young girls made morbid by the knowledge of a slight version or flexion, as if the uterus

were any more likely to be straight than is the nose, or more accurately adjusted as to position than are the media of the eye as to refraction. I have just returned from seeing a case of far-advanced locomotor ataxia with absolute blindness from atrophy of the optic nerves, the entire treatment of which, to date, has been gynæcological at the hands of professors in the foremost allopathic colleges in the country. Let me recount cases arising from injudicious local measures. A so-called estimable woman, worrying over her single fall from virtue, became nervous. Her physician discovered the cause in a certain uterine cervical disease which he promptly treated. This served only the more to intensify her worry, and direct attention still more strongly to the organs which she had misused. She became insane. A young girl suffering from melancholia made a very rapid improvement under proper moral and medicinal treatment. Her physician, yielding to the solicitations and superstitions of lay scientific(?) friends, began an investigation of the genitals, and discovering a uterine displacement instituted local treatment for the same. Immediately the poor child's attention was directed to a portion of the body of which she had hitherto been in ignorance, and her suffering mind once more broke down. Removal from home to a hospital became necessary. A complete cure without local treatment ensued. My gynæcological friends will tell me that the character of my practice is such that I see only their failures. I can retort that I see many of their successes so-called, successes which from a neurological standpoint are rank failures; cases still persisting in the delusions of their physicians, namely that the beginning and end of women's ills are the uterus and ovaries. I can on the other hand point to successful treatment of hysterical patients presenting marked utero-ovarian symptoms without other than constitutional and moral treatment. On this subject no men are better qualified to judge than Goodell and Playfair—both of them men widely and favorably known as practical gynæcologists, highly educated in general medicine, and of sufficient force of character to acknowledge the errors of the past and teach principles inculcating advanced methods. What is more surprising is that the superstition at which I have been inveighing has been so largely taken up by our homœopathic gynæcologists, indeed by homœopathic physicians generally. The very principles of our school provide for constitutional predisposition as the cause of very many local disorders, and yet they run to the other extreme of finding a local cause for constitutional troubles, too often seeking the same in the uterus. Those afflicted with special forms of mental astigmatism are just as liable to find the cause in the eyes, ears, nose, throat, heart, etc. Truly, false specialism has run mad in the land!

While thus decrying against indiscriminate special treatment in the hands of injudicious gynæcologists, oculists and others, I do not wish it to be understood that I advocate the other extreme, that of avoiding treatment

directed to special organs. Whenever the uterus, ovaries, eyes, stomach or other viscera show objective lesions, *i. e.*, conditions other than neurotic, they should receive careful attention. At the same time such attention should be given with due care, lest the patient be made morbid in that particular direction.

Non-gratification of the sexual appetite has been assigned as a cause of hysteria. It is probably operative only in amorous young widows, and married women with impotent husbands. This cause must be a rare one, for unless the patient be highly neurotic, the desire can be conquered. The peculiar neurotic condition of old maids finds other causes than want of indulgence, causes dependent upon the absence of affection and family ties—so necessary to the complete life of a woman's nature. Excessive sexual indulgence is far more apt to be a cause of hysteria and the allied neuroses.

Hysteria sometimes occurs epidemically by imitation in communities and schools.

Organic diseases of the nervous system as of other organs very often awaken hysterical symptoms in the predisposed. Thus strange combinations of organic and functional symptoms arise, making the recognition of the true nature of the complaint a difficult one.

**Symptomatology.**—The kaleidoscopic variety in which the symptoms of hysteria arrange themselves, makes a description of the clinical course of the disease highly unsatisfactory. It can be said of hysteria more truly than of any other disease, that hardly two cases are alike. Symptoms vary according to the peculiarities of the individual. Hardly an organic disease of any organ exists that is not capable of close simulation by hysteria—a simulation at times so accurate that the keenest clinician reaches a correct conclusion only after continued observation.

For convenience the symptoms of hysteria have been divided into the paroxysmal and interparoxysmal. Yet, strictly speaking, all of the symptoms to be hereafter detailed may come under the former heading, for they are liable to appear and disappear with remarkable fickleness. Prominent among the interparoxysmal symptoms, and upon which we are accustomed to rely for diagnostic purposes, is the mental condition. This is characteristic. It consists of a morbid self-consciousness, an emotional instability, a selfishness, a tendency to magnify and dwell upon every real and fancied ill. The patient exhibits a most intense egotism. Her friends and relatives are forced—sometimes in their irrational concern over the invalid's welfare (?) they are willingly led—to become her veritable slaves. The hysterical temperament may not be apparent to superficial observation, indeed, it may be completely subordinated to a disposition but little short of angelic, in the presence of strangers, to be converted into pure devilishness in the privacy of the family circle. Frequently it is exhibited by a marvellous attention to detail in the narra-



tion of symptoms—in the description of sufferings qualified by superlative adjectives—while the facial expression and the general manner give the direct lie to the words. She is very apt to regard her sufferings as the most intense, the most peculiar, the longest lasting of all invalids. She is the most sensitive of all persons to external influences. She cannot exert herself, she cannot partake of a favorite dish, she cannot be exposed to draft or cold, but that she suffers in consequence. Her will is weak. She is entirely unwilling to exert herself to acts which do not precisely suit her whims or fancies. The love for sympathy leads to unintentional exaggeration, and even to the creation of imaginary ailments. Sometimes not intentional simulation, but fears and ideas call the symptoms into being. The mere act of thinking of a dyspepsia, a neuralgia, a heart disease, a pneumonia, or any acute or chronic affection, is sufficient to call its counterfeit into existence. These mental symptoms are not always present to the extreme degree indicated by the above description. They exhibit all grades of mildness and severity, even in the same patient. The emotional temperament in which the patient alternately laughs and cries with insufficient or no reason is not as frequently present as one is generally lead to believe. Sometimes it is manifested mainly by an entirely inappropriate kittenish disposition. It is not contended that the outrageous behavior of the hysterical is the product of an innate depravity. It is on the other hand the product of diseased mental action. The patient suffers; she is truly far more unhappy than is the victim of a known incurable affection.

In very few cases of hysteria are sensory symptoms absent. Usually these partake of the character of par- and hyperæsthesia, less frequently of anæsthesia. To judge from literature, one would expect the anæsthesia to be the most common. Hyperæsthesia in hysteria is very irregular in its distribution. It may be widespread, or it may be strictly localized; or it may occur in patches in the midst of anæsthetic areas. It may be localized in the genital organs, leading to painful coition or vaginismus. Sometimes it affects the special senses, giving rise to photophobia, dysacusis, etc. The sensitiveness to touch is nearly always superficial, deep pressure causing, as a rule, but little if any discomfort.

The photophobia from retinal hyperæsthesia is generally treated by sympathetic friends with dark glasses, a means of relief that almost inevitably tends to the aggravation of the condition. This symptom is by no means uncommon. It may exist in a given case as the sole hysterical manifestation. Sometimes the sensitiveness is only to special colors. Before the hysterical paroxysms, visual hallucinations may take place.

The disturbances of taste and smell are sometimes of a most peculiar character. Thus the hysterical woman may develop a fondness for odors and flavors that disgust the ordinary person, while she

affects a repugnance to others generally regarded as of a highly agreeable nature.

The hysterical woman abounds in pains, and in every portion of the body. These may be spontaneous, or they may appear only in response to certain stimuli. Nearly always they are described as neuralgic, though they rarely follow the course of any nerve. They may be referred to muscles, fascia, bones or internal viscera, and may thus simulate quite a variety of affections of these parts. The patient uses the most extravagant language in describing them. With a calmness suggestive of the greatest self-sacrifice, she speaks of them as "mortal," "agonizing," "terrific," etc.

More or less headache is complained of. The characteristic hysterical headache is that generally known as the *clavus hystericus*. The pain is sharply defined, and limited to a very small portion of the scalp. It is very severe, and is apt to be excited by emotional influences or the menstrual period. It is often accompanied by nausea and vomiting, and subsides with the excretion of a large quantity of limpid pale urine. Almost as frequently one meets with hysterical headaches of a very different character. The pains are more or less constant. They partake of the features of hysterical complaints generally. Such patients usually go the rounds of the oculist and other specialists, fail to find relief, and finally under neglect recover or develop the full-fledged hysterical state.

Hyperæsthesia of mucous membranes sometimes exists. Affecting the alimentary tract, the greatest possible variety of digestive symptoms present themselves. Involving the laryngeal mucous membrane, there develops a sensitiveness to any unusual temperature, or to particles of dust floating in the atmosphere, as shown by paroxysms of coughing.

Neuralgic pain in the mammary gland—*mastodynia*—is not infrequent in hysteria. The pain does not seem to be any different from that encountered in similar cases arising from anæmia. Other thoracic pains are those like intercostal neuralgia, and a peculiar heavy feeling beneath the sternum. The latter is of especially frequent occurrence, and is the cause of great sense of oppression and anxiety. If accompanied by disturbed cardiac rhythm, apprehensions of heart disease become a source of suffering.

Hysterical pain may affect any or all portions of the abdomen. Widely diffused and associated with great hyperæsthesia, it leads altogether too frequently to the diagnosis of peritonitis. Sometimes the pain locates itself in the epigastrium, and simulates gastralgia or gastric ulcer. Especially is the latter affection suggestive, when vomiting is profuse. Ovarian pain is very common. It is usually left-sided, is aggravated by pressure, and may or may not be accompanied by actual disease of these organs. It must not be regarded as anything else than a hysterical manifestation.

Pain in the back is present in practically every hysterical patient. Usually, there is great sensitiveness to touch, limited to particular regions of the spine. Sometimes the pain is spontaneous, and sometimes it occurs only when evoked by pressure; it may be limited to the spinous processes, or it may be diffused laterally into the muscles. This condition has, for a long time, been referred to as spinal irritation, and incorrectly attributed to spinal anæmia.

Hysterical pain may affect various joints. It occurs with greatest frequency, however, in the hip and knee. There is pain from pressure on the affected joint, but it seems to find its origin in the soft parts rather than in the joint structures themselves. The symptoms vary greatly in intensity from day to day. It has been suggested that some cases of vertebral pain are local examples of hysterical joint. Swelling, when present, is limited to the soft parts.

Anæsthesia does not seem to be as frequent a symptom of hysteria in this country as it is in Europe. It may very readily escape attention unless searched for. It may assume almost any distribution. It may be limited to one lateral half of the body—hemianæsthesia—or it may affect one organ. The anæsthesia may occur spontaneously, or it may follow a convulsion. It very frequently involves the sense of sight, giving rise to amblyopia, hemianopsia, concentric limitation of the visual fields, and achromatopsia.

Paræsthesiæ exist, in the vast majority of cases, in every possible variety. This and that morbid sensation occurs in almost any and every portion of the body. Sometimes they are simply annoying by their presence; at other times they excite the wildest apprehensions of the patient, lest some organic disease of an important viscus be present. In many instances they consist of hot flashes or cold chills, while in others they take a more specialized form. Sometimes they are such as to possess the patient of the idea that this or that organ (generally the intestines) has ceased to perform its function, a false belief that persists despite the most positive objective evidence to the contrary.

The loss of sensation is not always alike in different cases. Sometimes it affects the touch, sometimes the temperature, and sometimes the pain sense. It is possible for the latter to be so completely obtunded that the patient can perform severe self-mutilations without apparent suffering. Many authorities believe that this absence of pain under such circumstances is assumed, and that the mutilations are performed out of pure love of exciting the interest of others. The anæsthesia may also involve joints and muscles and mucous membranes. Affecting the genital organs, complete loss of sexual desire occurs; and involving the bladder, it leads to retention of urine.

Much interest in the phenomena of transfer, as applied to the sensory disturbances of hysteria, was excited a number of years ago. It



was first shown that under the influence of magnets or disks of certain metals, the hyperæsthesia, etc., could be made to change from one part of the body to another. It was soon proven that these transfers were the direct result of psychic influences, for they took place under the supposed action of sham magnets and metals as readily as with the genuine.

Hysterical paralyses may assume the type of paraplegia, hemiplegia or monoplegia. They almost always involve the extremities, and almost never the muscles tributary to the cranial nerves. Facial and ocular palsies are said by Wood to be of occasional incidence in hysteria. This statement, if hysterical ptosis be omitted, lacks decidedly, in my opinion, of clinical confirmation. Paralysis of the tongue leading to difficult articulation, though very rare, is doubtless possible. We are accustomed to diagnose the trouble of hysterical origin, when a paralysis appears in a person presenting undoubted symptoms of hysteria. But care must be taken to determine that such palsy is not of the usual organic types, occurring in a patient already ill with hysteria.

Hysterical paralysis of any type may be of either rapid or slow onset. Usually, it appears suddenly following a convulsion, or in response to some violent emotion. It may simulate any type of organic palsy. In the majority of cases the lower extremities suffer the most. The loss of power is almost never absolute. There at first appears a sudden, partial loss of power, which gradually increases in severity and extent. The legs can be moved more or less freely while in bed, but, on attempting to stand, they exhibit a helplessness, disproportionate to their activity at other times. The paralyzed muscles rarely undergo any atrophy at all extensive. They may become flabby from malnutrition or non-use. Their electrical reactions are invariably normal. The deep reflexes are usually increased, or normal. Wood says they may be destroyed. Herein I think he is again in error. The same authority agrees with certain other prominent neurologists in permitting of a well-marked ankle-clonus as consistent with hysteria. This again is probably a mistake, arising, doubtless, from the frequency with which disseminated sclerosis in young women is confounded with hysteria. The only ankle-clonus occurring in hysteria is the spurious ankle-clonus described by Gowers. "When the foot is first pressed back, there is no movement, but presently a contraction of the calf-muscles causes slight extension, and with it the clonus occurs. This spurious clonus varies from moment to moment, sometimes almost ceasing, and is again renewed by a fresh contraction of the muscles." When the knee-jerk is found absent, proper examination will disclose the fact that such absence is due to contraction of the hamstring muscles, a condition readily recognizable by palpation of these tendons, when the knee-jerk is sought for.

In hysterical hemiplegia the leg will be found more powerless than the arm. The superficial reflexes unlike those of organic hemiplegia, will be found exaggerated or normal, never diminished.

Sometimes an ataxia of movement is observed. This is due to muscular anæsthesia, and is absent when the patient is in bed, but prominent enough when she is up and about.

Local and visceral palsies are sometimes prominent features. Of these hysterical aphonia is the most common, and has found thorough elucidation in the chapter devoted to laryngeal paralyses. Swallowing is sometimes rendered difficult or impossible by paralysis of the muscles of deglutition, making the use of the stomach tube for feeding purposes necessary—a measure that not infrequently acts curatively with remarkable promptness. Paralyses of the bladder and bowels are sometimes observed.

The spasmodic manifestations in hysteria are as varied in character as are the paralytic. They may appear as contractures or as convulsive seizures. The latter may again consist of tonic or clonic spasms or of co-ordinated movements. Whatever the character of the spasms, they may invade any or all portions of the body under any and all circumstances, and in the most varied fashion. Taking first the contractures, we find these to be a condition in which the affected part is fixed in a state of tonic spasm. They may come on suddenly or gradually; after a convulsion or injury, or violent emotion or spontaneously; and they may last but a short time, or they may continue for months; and when they disappear, they may do so suddenly with or without apparent cause. While the spasm lasts, the rigidity is usually the same from day to day; but not infrequently, this too presents great variations in intensity. It is increased by any attempt to overcome it. It often persists during sleep. The arms are more frequently involved than are the legs, the position being one of flexion at the different joints, thus simulating the late rigidity of hemiplegia. In one particular it differs from the latter, however; when passive flexion is performed at the wrist joint, the flexion of the fingers is not thereby lessened.

Hysterical contracture sometimes comes on after traumatism. It may then appear either slowly or suddenly, usually the latter. Such cases are often mistaken for neuritis, but differ from the latter in that hysterical contracture very often spreads to other extremities than the one in which it started. Anæsthesia exists to some degree in nearly all cases in the limbs whose functions are disturbed. When the leg is affected, the position is one of extension, the feet often being on a line with the legs, with the toes flexed and the feet inverted. Usually but one leg is affected, contractures of both lower extremities being very rare. From long-continued contracture, muscles may undergo organic alteration, the shortening becoming permanent. Cases have even been reported in which the spinal cord itself has become diseased. Such instances are, however, rare, and are liable to the interpretation of mistaken diagnosis, the cases probably having been organic spinal disease from the start.

The contractures of hysteria are occasionally limited to portions of muscles, thus giving rise to the impression that a tumor exists. It has been suggested that some cases of phantom tumor of the abdomen are thus occasioned, though Gowers offers as an explanation of their appearance, a spasmodic contraction of the diaphragm, and relaxation of the recti muscles.

Simple tremor, especially as the result of excitement, is quite common in hysteria. Sometimes, however, it occurs in paroxysms resembling general clonic spasms. The movements may then be universal and continue for hours, and even for days, as in one case under my treatment. Hysterical tremor belongs to the group of intention tremors, and may thus be confounded with that of disseminated sclerosis, but is readily distinguished from the latter affection by its regularity of rhythm, and the absence of inco-ordination. It may locate itself in any portion of the body, and is very apt to appear or become aggravated under very slight emotional influences. It may be checked by peculiar agencies. Thus I have seen it disappear promptly on making firm pressure over the active muscles, their nerves, or other portions of the body, and by diverting the patient's mind from her sufferings.

The convulsive seizures of hysteria consist of tonic or clonic spasms, and co-ordinated movements, one or all. In their mildest form, they may appear only as slight rigidity following possibly some emotion, unpleasant or otherwise. In other cases they are manifested only by "*globus hystericus*," or a few purposive movements. While ushered in suddenly, yet the patient never falls so as to hurt herself. She manages to reach a place of safety, or lets herself down easily. Sometimes, however, by accident, she does injure herself. Some degree of apparent unconsciousness is nearly always present, but it is readily seen that this is not profound. Possibly some few clonic movements may appear. In still other cases the patient simply clutches at her clothing and surrounding objects.

The severe paroxysms of hysteria are usually referred to as *hystero-epilepsy*. They are seen in their best developed form in France, rarely in England and this country. The positions assumed by the patient are both grotesque and theatrical. At one moment bent in a position of extreme *opisthotonos*, the body resting on the forehead and toes, and at another making the wildest bounding movements; now assuming expressions of pleasure, and the next of extreme depression or terror; now exhibiting religious fervor and then becoming erotic or obscene; now rolling about the bed and then lying in perfect quiet; a picture certainly alarming to the uninitiated.

The severe paroxysms, as seen in America, are often ushered in by an aura, consisting usually of the *globus hystericus*, palpitation of the heart, or the bilateral foot aura. They may appear spontaneously, or as



the result of some violent emotion. They are often ushered in by a loud cry, which very often continues throughout the paroxysm. This cry may consist of a mere shriek, or of words appropriate to some emotional state. Sometimes the patient talks considerably during the paroxysm, the words generally being mumbled, and without any connected meaning. Opisthotonos is very often present. It not infrequently appears with the patient lying on her side.

The clonic spasm of hysteria resembles that of epilepsy very closely, but differs from that of the last-mentioned disease in not being so shock-like in character, and in maintaining the same frequency throughout its duration. In epilepsy the clonic spasms maintain the same strength throughout, but towards the end, as the spasm is disappearing, they occur at longer and longer intervals. Sometimes the hysterical clonic spasm consists of but a coarse tremor, or is limited to unusual portions of the body, *e. g.*, both hands or both feet.

The distinctive movements in hysterical convulsions are the co-ordinated. These simulate voluntary movements. In a general way they may be described as "struggling." The patient squirms, stretches, strikes, clutches at her hair, tears at her clothing, bites herself and others, kicks, strikes the head against the floor, etc. These movements are repeated with the utmost irregularity. Exceptionally, one or two movements are selected and repeated rhythmically. It is not unnatural that these attacks should terrify the bystander, and lead to strong efforts to restrain the patient—efforts which almost invariably only intensify the convulsions. Almost certainly can we rely upon it when we are told that it required several persons to control the patient because of the violence of the attacks, that the convulsion was hysterical in character.

The mental state in hysterical attacks is as varied as are all the other symptoms. Frequently the patient is merely emotional. More marked departures from the normal mental condition may be present, and consist of terrifying hallucinations, ecstasy, and even a condition closely simulating mania. This brings up the question of the relationship of hysteria to insanity. It is quite common in practice to meet with mental cases in which hysteria has been diagnosed by the attending physician. In the great majority of such, this is a distinct error. Undoubtedly most of these patients are hysterical to some degree, but it must be remembered that hysteria can engraft itself on insanity, as it can on any organic disease of the nervous system.

Visceral spasms sometimes form the prominent source of complaint in hysteria. The globus hystericus constitutes the most frequently observed of these. Sometimes this is substituted by a sensation of constriction, or by the feeling as of a foreign body in the throat. Spasm sometimes invades the œsophagus, and makes itself known by regurgi-

tation of food before it has time to reach the stomach. The stomach may be involved, so that vomiting of various grades of severity appears. This may occur at long intervals, or be so persistent as to seriously threaten the patient's welfare. Food may be ejected as soon as it enters the stomach. The malnutrition following this hysterical vomiting is not as great as the seriousness of the symptom would lead us to expect. This is probably owing to the fact that despite appearances some of the food is retained and assimilated. Hysterical patients sometimes speak of a persistent vomiting during which only mucus is ejected. Such cases seem to me to hardly deserve the name of vomiting. It gives the appearance very much of quasi-voluntary retching movements. Nutrition is not disturbed by them in the least.

Retention of urine is sometimes of spasmodic origin. It occurs either with or without associated painful disease of the genito-urinary system. Urging to urinate may be frequent. The introduction of the catheter meets with resistance.

Hysterical cries sometimes exist as almost the sole symptom of a case, though just as often they are associated with other phenomena described in this article. They nearly always imitate the sounds of certain animals. Thus the patient at regular intervals gives forth the sound of a cuckoo or barks in imitation of a dog. These phenomena are generally spoken of as beast mimicry. All of the cases of spurious hydrophobia, in which the patient bites at attendants and barks, are of this nature.

Hysterical visceral symptoms are possible in connection with any organ of the body. Those of the respiratory system are quite common. One of the most frequently noted of these is a peculiar disturbance in the respiratory rhythm. After a number of normal respirations there occurs one longer drawn and deeper than the others. This often occurs from unusual nerve wear and tear or from undue worry. Another type of hysterical breathing is found in dyspnœa. This occasions no especial amount of distress, and is not associated with any disturbance in the frequency of the pulse. Still other cases present simply rapid breathing. The respirations are increased in frequency in most cases of hysteria; but reference is here had to cases in which the increase in the rhythm is something remarkable, *i. e.*, ranging from 120 to 150 per minute. There are cases in which the hysterical dyspnœa presents rather alarming objective features. The patient sometimes becomes cyanosed in such. Even laryngeal spasm may present itself. An hysterical cough has been described. It is usually of dry, persistent character. Hysterical hiccough rarely counterfeits this symptom as ordinarily observed. It is, however, none the less annoying and distressing to attendants.

Of the hysterical affections of the stomach, vomiting has already been mentioned. These patients sometimes exhibit remarkable perversions

of the appetite and even absolute anorexia, in which there is disgust for all food. There may be exhibited an inclination to partake of the most outrageously indigestible food, or to refuse food of all character for weeks. Many cases with gastric disturbance present considerable flatulency as a prominent symptom. This occurs irrespective of the character of the food taken.

Either constipation or diarrhœa may occur as a purely neurotic symptom. The former is often obstinate, requiring for its relief the most painstaking care. Its hysterical character is shown by its prompt disappearance many times under treatment by simple isolation. The neurotic diarrhœa, like other hysterical symptoms, occurs under emotional influences.

The cardiac symptoms are very common, and often give rise to the most absurd fears of organic heart disease. They consist generally of excited action from slight emotions with pains in the cardiac region. These paroxysms may even be accompanied by fainting or dyspnœa.

Of the renal symptoms, hysterical polyuria is the most frequent. Especially after an attack large quantities of pale, watery urine are voided. The fluid in this abnormal condition seems to have the power of producing vesical irritation, leading to frequent calls to micturition. The polyuria of hysteria is explained by dilatation of the renal blood-vessels, which takes place under emotional disturbance. Very rarely anuria occurs. This symptom, when of hysterical origin, is not attended by the phenomena of uræmia, and may last for a number of days without serious result. It is then almost always associated with vomiting.

Vaso-motor symptoms, consisting of morbid local flushings and pallor, and inordinate local and general sweating are frequently observed. Very few cases of hysteria fail to exhibit these phenomena.

Hysterical hæmorrhages and hysterical pyrexia are almost invariably examples of deception or mistaken diagnosis. In the case of the latter, the thermometer often registers a very high point, 110° or higher. In one such case under my observation, the high temperature was secured by the placing of the thermometer against hot water bottles when the nurse's back was turned. Remarkable cases of hysterical hæmorrhage grow less and less frequent as clinical medicine advances, so that we are justified in believing that all cases reported in years long since are to be relegated to the class of arrant frauds.

**Diagnosis.** — Ordinarily the diagnosis of hysteria meets with no difficulties. Cases will arise, however, in which all the resources of the most astute clinician will be called into requisition. In such a complete command of all the means of physical diagnosis is the only safeguard against error. There is hardly a disease of any viscus which may not be simulated. Differentiation is then only possible by a process of exclusion. The diagnosis is thus made on negative data. Not unnaturally



cases in which hysteria is diagnosed become less frequent as skilful technique increases. As affirmative evidence such symptoms as the globus and the peculiar mental condition of hysteria are valuable. Sight must never be lost of the age and sex of the patient as diagnostic factors. The remarkable variations exhibited from day to day by hysterical cases must always be borne in mind, and accorded their full value. The readiness with which symptoms disappear in the presence of strangers, and become aggravated in the midst of undue sympathy, is of importance. Sometimes the present symptoms so closely simulate those of an organic affection that a diagnosis based on them alone is impossible. Then one must have recourse to other illnesses—in other words, to the history of the patient. The recognition of a single symptom positively indicative of organic lesion is of the utmost value, as it outweighs in value a host of hysterical phenomena, for, as already stated, it is by no means unusual for organic affection of any viscus to produce hysteria in the predisposed. Of organic nervous affections most liable to be confounded with hysteria, the most common source of error is disseminated sclerosis, an error arising from the occurrence of the latter disease in young women, and the remarkable variations it presents in symptoms from time to time. The discovery of symptoms positively significant of structural changes makes the diagnosis clear.

**Prognosis.**—This must be considered from the double standpoint of life and recovery. Very few cases of hysteria have a fatal termination. Even the vast majority of cases of severe hysterical vomiting recover. Some few die.

To state the prospects of recovery in any individual case is always a difficult matter. It must be remembered that any case of hysteria, however severe, may recover. On the other hand, one must also bear in mind that these patients are ever surrounded by the depraved influences to which the affection originally owes its birth—faulty home influences and a neuropathic constitution. The nervous system forms habits as readily as do individuals, and bad habits once formed always afford a difficult subject of cure. To secure the full degree of success, one must control not only the patient, but also a large circle of friends and the entire home circle. The reference to “habit” predetermines that the older the case the greater will be the difficulties to be encountered and to be overcome. But again, even old cases may make very unexpected recoveries, sometimes spontaneously, sometimes under treatment, and sometimes under the influence of accidental emotions. Paralyzes and convulsions nearly always recover. Contractures offer a more difficult problem. Confirmed hystero-epileptics often lead a life of permanent invalidism. Some patients are so wedded to their woes, that they would not part with them if they could. Of such the least said the better.

**Treatment.**—From all that has been said, the reader will at once

rightly infer that there is much in the proper treatment of hysteria, and very little in medicine, at least in the management of aggravated cases, in which the neurotic element is predominant. The treatment resolves itself almost exclusively into measures of a moral character, and those designed to elevate the standard of bodily nutrition. This remark is alike applicable to both the prevention and the cure of the disease.

Preventive treatment, to be efficient, should be early begun in those predisposed. It involves the employment of all measures increasing bodily health; in other words, the leading of a hygienic life; but here one is in danger of running to an extreme, especially if the near relatives are, as nearly always happens to be the case, morbidly solicitous concerning the many little woes of which the patient complains. Extravagant attention to these only serves to intensify them. Again, the nervous system should be educated out of its morbid sensitiveness to every slight external impression. This may be done by the avoidance of enervating influences, as too luxurious and aimless a life, and the adoption of a course involving moderate exposure. Here, again, another extreme stares us in the face, for by too much exposure, too hard a life, hysterical troubles may be brought on. The principal point, however, is found in the education of the child, so that a career of self-control and usefulness to self and others is learned.

The curative treatment depends largely upon the extent to which the disease has developed, and the unwholesome character of the patient's surroundings. It largely resolves itself into measures of a general character, consisting of a judicious combination of isolation, rest, overfeeding, massage, and electricity. Put in this way, the problem is apparently an easy one. It must be remembered, however, that each case requires the most careful individualization and study to determine to what extent each of these measures must be enforced, in order to make their combination a judicious one. In addition to this, individual symptoms, arising from complicating diseases and dyscrasiæ, must be treated in many instances. To treat all cases as dependent upon some reflex cause, to give a nurse, to isolate completely, to enforce absolute rest, to stuff with food, and to manipulate *ad libitum*, is gross empiricism, and the height of therapeutic folly. To adapt each of these to the case in hand, means almost certain success.

In all cases, mild or severe, the personal qualities of the physician constitute a very important factor. Some physicians possessed of the highest scientific ability are so decidedly lacking in these qualities as to make success with them exceedingly doubtful. Others less skilful, by reason of their possessions achieve successful results with but little labor. Some possess these qualities by nature; others acquire them; and still others are so deficient, as to ever remain failures. Tact, firmness and positiveness on the part of the physician are ever necessary. He must

exhibit a confidence in his resources to imbue his patient with confidence in him. He must be unflinchingly firm in his demands that his directions be carried out. It is sometimes necessary that he be actually cross, but with all he must ever be a gentleman. To scold and rail at the patient because of her wails and woes rarely accomplishes one iota of good. Sometimes by his intuitive knowledge of human nature, the physician is enabled to reach the patient by awakening an emotion to arouse her from her life of selfish invalidism. To illustrate the effects of emotional agencies in the care of such patients, I may relate a case, counterparts of which are doubtless familiar to all. A lady had been for years a bed-ridden invalid. Physicians had been in attendance for years without giving the desired relief. Her faithful husband was finally taken dangerously ill with an acute fever. Immediately she was enabled to arise from her bed; she nursed him through a dangerous illness, and on his recovery she remained well. A severe fright has cured other cases; and still others have recovered from wounded pride. To utilize the foibles of female human nature for therapeutic purposes requires a rare combination of skill and tact.

The treatment of special symptoms in hysterical patients must be carried out with care lest it be overdone. As a rule, when such symptoms are of purely nervous origin, they are better ignored. When dependent upon complicating factors, attention to them is necessary. Even then the general condition of the patient must be borne in mind. Any actual local trouble should be removed. When, depends not only upon the case, but also upon the plan of treatment proposed. As a rule, unless the inconvenience produced by them is very obtrusive, they had better be let alone until the general state of health has improved, for it will very often be found that they disappear spontaneously as it were, under constitutional treatment. Especially is this advice applicable in severe cases in which nervous prostration is extreme. Above all things radical surgical operations, whether in the domain of general, orificial or gynecological surgery, should be postponed. And again they should never be done unless there are distinct objective or subjective phenomena arising from them, and which would *per se* indicate the operation, were the nervous manifestations absent. It has been claimed for orificial surgery that the best results are attained in cases in which local lesions have given rise to no direct symptoms. It is more than probable in such the local lesions have been small or mythical, and the operation for their relief correspondingly mild and involving no surgical shock. Consequently the patient gets the moral effect of a surgical operation and the rest during convalescence from the same.

Any and all conditions involving waste, need prompt attention. Hæmorrhages of all kinds must be controlled promptly. Vomiting must be checked. The most efficient agent for this latter symptom is the stomach tube. Sometimes, however, it fails.



It is unwise to resort too early to the catheter in hysterical retention of urine, as patients soon become accustomed to it, and demand its repeated introduction.

It is bad practice to treat hysterical spines by supports and orthopædic apparatus of various sorts. They sometimes do good by the psychical influence they exert, though they almost invariably intensify the very trouble they were designed to relieve.

In cases of morbid sensitiveness of the eyes and ears, it is not advisable to cater to the symptom by ordering dark glasses and cutting off all sounds. The sensory hyperæsthesia is not lessened thereby. On the contrary, it is nearly always increased. When patients are found thus treated, it is well to withdraw the glasses in the one case, and the dampening of sounds in the other. Such advice may appear cruel, but it is wholesome.

Convulsions are best treated by means making a powerful physical impression. Neglect of attendants is here sometimes valuable. Sometimes the attacks may be checked by pouring ice-cold water over the patient, or by irritating the fauces. In severe cases, the hypodermic administration of from one-tenth to one-twentieth of a grain of apomorphia, as recommended by Gowers, brings the contortions to an end. This drug produces vomiting within a very few seconds after its administration, and, with the onset of the vomiting, the spasmodic symptoms subside.

Hysterical pains are best treated by teaching the patient to disregard them. Give her to understand distinctly that they possess no significance aside from the discomfort they cause her, and that it is best to go about her duties, regardless of their presence. This may be a difficult, a well-nigh impossible task at first, but if the patient is taught perseverance, she will conquer them.

Sexual indulgence is by no means necessary as a therapeutic agent. Illegal gratification of the sexual appetite should never be countenanced by physicians, for a life of continence is consistent with health. The advice to get married must be looked upon with disfavor, unless a companion with whom the patient can live in love and happiness, has been already selected. As one writer, whose name I cannot just now recall, has happily put it, a wife or a husband must be regarded from a higher standpoint than a therapeutic agent.

Sexual excesses are far more liable to do harm. In fact, if one is to err in giving advice on this point, it should be on the side of a high standard of morals, and counsel moderation rather than overindulgence. It may happen that the frequency of indulgence is not at fault, but the manner of gratification. There are many ways of violating sexual hygiene.

The treatment of hysteria by seclusion, rest, overfeeding, massage

and electricity was first proposed by Weir Mitchell a number of years ago, and is often spoken of as the "Weir Mitchell treatment." As ordinarily understood by those not using it, it consists of a number of hard and fast rules to be applied in routine manner to every case. As practised successfully it includes a judicious combination of these various agencies. Even the selection of the nurse is a very important matter, for the best directed efforts can be rendered futile, if the attendant is not adapted to the case in hand. Speaking on this point, Mitchell observes: "In choosing a nurse remember that if she has no tact or has a short temper, or is clumsy, or unneat, you may have your case spoiled, or be forced to change the nurse midway in your treatment; but at all events never hesitate in this. If the patient and nurse do not agree, make a change, and if need be, another. I cannot enough emphasize this matter of the nurse. Put yourself in the place of an intelligent lady shut up for two months with a coarse woman, whose talk and habits disgust, and doubly disgust, because the victim is emotional and sensitive by nature and habit, and you will realize the need for care in your choice of an attendant. Mere technical training will not answer, and I have seen an utterly untrained woman of good brains and tact win successes which are sometimes denied to the best educated nurses, who lacked those ever-needed moral qualities which no training and no length of experience will give to some women."

Isolation is necessary to some extent in all cases. In severe ones, it must be carried to the degree of removing the patient from all friends and relatives. In others, it is only advisable to remove her from certain ones whose methods and actions tend to intensify the hysterical habit. These parties are often the ones whose companionship is most desired by the patient. Even the receiving and writing of letters is dangerous. It is this isolation to which patient and relatives most strenuously object when the "rest treatment" is proposed as the one measure for their cure. It involves nearly always removal from home and the breaking up of accustomed associations. In some few cases, residence at a resort in company with a proper nurse to supervise and direct her daily life untrammelled by the criticisms of anxious relatives is all that is needed. In still others, when the hysterical element is not predominant and the neurasthenic features are well defined, rest at home may prove successful. When removal from home is decided upon, the character of the place selected is important. It should never, when it can be avoided, be a general hospital, for the presence of other patients, the frequent surgical operations, and the odor of antiseptics and ether generally work injury. It should be a boarding-house or establishment arranged for the reception of just this class of cases. Sanitariums are rarely advisable, for so far as my observations go, they are generally managed so as to foster attention to minor ills of the neurotic, and in the end intensify hysteria.

Patients are too apt to get together and hold converse over their ailments—an evil altogether too common in every community—and this must have a bad influence. All rest establishments are not equally well conducted. Those in which the management looks more and more to home comforts and homelike surroundings, and less to the atmosphere of the hospital or sanitarium, should be chosen when available.

The next element in the treatment is rest. In most marked cases this should be absolute at first. The patient should be kept in bed forbidden to exert herself excepting to the extent necessary to perform the necessities of life. It may indeed prove desirable to forbid her rising for any reason whatever, compelling her to use the bed-pan for stool and urine, and have her fed by the nurse. These severe cases require usually a course of from six weeks to two months in bed. When the time to permit them to get up arrives, it should be by degrees. At first they should sit up in bed during meals; then they should sit up each day long enough to have the bedding changed; and finally permitted to remain up for fifteen minutes three or four times daily. Then walking should be attempted, small efforts being made at first, increased in duration from day to day.

Some cases require a restricted period of rest from the beginning. These include cases in which the love for the couch or the bed is already too great, and in whom a course of absolute rest will be regarded with altogether too much satisfaction. Other cases are not sufficiently ill to require the absolute rest. In these cases, the best plan is to order the patient to remain in bed until an hour or so after breakfast; and to take one hour each of absolute rest in bed after dinner and after supper. This course should be followed for some time after convalescence has been established. In fact it is a question if these nervous invalids even when restored to health should not keep a certain hour each day sacred for rest purposes for all time to come.

As soon as possible the patient should be gotten out in the open air. At first it should be attempted in the carriage; later by walking. The time spent in these occupations should be fixed by schedule for each case. Even before sufficient strength to go out is gained, the beneficial effects of out-door air may be obtained by having the patient dressed for the street, at an open window, one exposed to the sun being preferred.

Massage should be performed, when possible, by a professional masseuse. The greatest care should be exercised in selecting one. She, as well as the nurse, must be a woman of tact and discretion. Going from house to house gives her the temptation and opportunity of spreading gossip—an evil habit that cannot be too severely condemned. She must avoid any conversation concerning the ills of the patient or of others; and she must avoid all discussion concerning the merits of the treatment. The best time for her visits is in the middle of the morning;



but as she cannot attend to all her cases at that hour, arrangements should be made for a regular time each day. To secure efficiency of service, unless the ability of the masseuse be known to the physician, the latter had better see her give one or two treatments. Playfair suggests, however, that one can judge best of the efficiency of her work by the way the patient takes food. If she eats and relishes the desired quantity, he is satisfied that all is going well. For economical reasons it is sometimes necessary to leave the manipulation to the nurse. The character of the massage in these cases is such that it can be readily performed by any intelligent healthy person. Usually, however, the nurse has enough to do in the management of the case without imposing this duty on her. The idea of massage is to secure the nutrition changes obtained by exercise without exposing the patient to exertion. At first there is sometimes some difficulty in the beginning. Patients think the manipulation does not agree. It is well to have the first sitting quite short, say twenty minutes. On successive days the treatments are lengthened until, finally, the patients get one full hour of massage daily.

The electrical treatment is carried out with a twofold object. First, for the relief of special symptoms, in which cases the ordinary rules of electro-therapeutics are used as guides; and, secondly, as an aid to massage, in giving the patient sufficient exercise. For the latter purpose the slowly interrupted faradic current is used, each muscle of the body in turn being treated, and made to contract several times. This electrical treatment may be omitted when circumstances do not favor it; that is, when the nurse is not skilled in the use of the battery. It rarely happens that the physician has sufficient time at his disposal to personally carry out the treatment.

Last, but by no means least, comes the diet. This should be conducted with the view of getting the patient to partake of as large quantities of food as possible. Beginning with the first days of the treatment, milk is occasionally made the sole article of diet, administering four ounces every three hours. Other patients are required from the beginning to take three full meals with a certain amount of nourishment generally in the form of milk between times. As to the articles which go to make up these meals and the luncheons, the fancies of the patient should be largely consulted, unless she is decidedly whimsical, and inclined to ask for outrageously improper food. With those patients who begin with an exclusive milk diet, solid food should be ordered in the course of three or four days, gradually increased in quantity until at the end of the first week, when three full meals are to be given. Some patients will declare positively that milk disagrees with them. In the vast majority of such, this idea is merely notional and nothing more. I have succeeded in assuring some of these that if the milk be thoroughly shaken, it will agree perfectly. The milk can be given if not in one form, then in

another. It may be given plain, mixed with Vichy water, with lime water, with bicarbonate of soda, or in the form of koumiss or peptonized milk. Liquid beef preparations are valuable aids to nutrition. The manufactured peptones, etc., are not to be thought of in this connection, but instead, home-made soups should be used. Of these the raw beef soup of Mitchell, or a beef extract made after the manner to be shortly described, is the best. To make raw beef soup one pound of good beef is chopped fine and mixed with a pint of water to which five drops of pure hydrochloric acid have been added. This is permitted to stand over night in the refrigerator. In the morning it is placed on the range and subjected to a heat of 110° for two hours. This quantity is to be divided into three portions and administered at appropriate periods during the day. It is of course seasoned to suit the taste. The flavor of the raw meat is sometimes objected to by the patient. In this case the meat is made into a ball and broiled superficially, after which it is subjected to the process already described. Another method of preparing a beef extract is as follows: A suitable piece of steak is broiled very superficially. It is then cut into small pieces, and put into a beef compressing apparatus. The juice thus extracted is administered after proper seasoning.

Constipation in patients taking large quantities of food is a serious matter, and must be avoided. In some cases, the taking of a cup of coffee the first thing in the morning without sugar or cream overcomes this difficulty. In others medicines are necessary. Nothing is better than aloin pills or cascara, used of course as a temporary expedient during the dieting.

The rest treatment as thus described usually takes up so much time that the monotony of life is avoided. Still opportunities will be found when the nurse should entertain the patient by reading to her. The character of the literature selected should be a matter of some thought. Trashy novels containing sensational matter should be avoided. Standard literature only should be chosen. Sometimes carefully selected reading from the daily papers is best. In selecting reading matter, the character of the patient's education and the tendency of her thoughts should be considered. Perhaps short stories by standard authors are the best suited to most patients.

As to medicine, very little is needed. Some patients require iron in physiological doses, preferably iron reduced by hydrogen, in doses of six grains daily. Malt extract at meals may be advisable.

Of the medicinal treatment of hysteria generally, but little can be said. Medication should be conducted with a view of improving the general condition, rather than of treating wild and largely fanciful symptoms. *Ignatia*, *pulsatilla*, *actea racemosa*, *nux vomica*, *belladonna*, and numerous other remedies of the materia medica may be necessary, but

they must always be looked upon as secondary in importance to the plan of treatment just outlined in these pages.

The daily schedule of the patient undergoing the rest treatment, as advocated by Mitchell, is as follows:

Cocoa at 7 A.M.

Cool sponge bath, with rough rub, and toilet for the day.

Breakfast at 8 A.M., with milk.

Rest an hour after.

8 oz. peptonized milk at 10 A.M.

Massage at 11 A.M.

Reading aloud by nurse, half hour.

Dinner at 1.30 P.M.

Rest an hour.

8 oz. peptonized milk at 3.30 P.M.

Electricity at 4 P.M.

Supper at 6.30 P.M., with milk.

Rest an hour.

Reading aloud by nurse, half an hour, 8 P.M.

Light rubbing by nurse with drip sheet at 9 P.M.

3 oz. malt extract with meals; tonic after meals.

8 oz. peptonized milk with biscuit at bedtime, and a glass of milk during the night if desired.

Laxative (cascara).

Later, Swedish movements are added after the massage.

This schedule must not be regarded as one fixed for all cases, nor, indeed, for every day for the one particular case. It must be regarded as a standard from which departures are to be made according to special demands of the case in hand.

Many cases cannot be removed from home for treatment. In such a course of treatment in which moral training enters largely should be followed. Especial care should be taken to prevent the patient's association with persons who have a manifestly bad effect on her and her illness.

In the practice of the rest treatment, the greatest judgment is required in determining the amount of rest and seclusion required. With some the simple act of placing the patient under the care of a proper nurse is all the exclusion needed; in others it is necessary only to send the patient away from home to some resort, where not too many invalids are congregated. There are cases, too, in which rest can be greatly abused, cases requiring activity instead of idleness.



## CHOREA.

The term chorea has been made to stand for quite a variety of spasmodic disorders. Originally it referred to one of an hysterical nature, a dancing mania of the Middle Ages. The superstition of the day provided a cure for the malady in a visit to the shrine of St. Vitus. Hence it was also called St. Vitus's dance. This latter name, as also the technical one, chorea, is now restricted so as to apply only to a disease occurring mostly in children and young people, characterized by irregular involuntary movements of the voluntary muscles, said movements occurring both during rest and attempts at motion, interfering with the accuracy of voluntary movements, and not infrequently attended by evidence of mental weakness.

**Etiology.**—*Age.* As intimated in the above definition of chorea, its subjects are practically all young. Fully 90 per cent. of the cases attack those between five and twenty years of age; and not less than 80 per cent. are between five and fifteen. There is, however, no age at which the disease may not occur, it having been observed at both extremes of life. The majority of cases, if we include only first attacks, occur about the eighth or tenth year; including all cases, the greatest frequency is probably reached at the age of puberty.

*Sex.* Girls are more frequently affected than boys, in the proportion of about two to one.

*Race.* The most reliable evidence shows that chorea is rarely met with in the pure-blooded negro.

*Heredity* undoubtedly is not without its influence. In about one sixth of all cases there is transmitted a neurotic constitution which predisposes to the disease. In other cases, heredity has been supposed to have proven an important etiological factor by reason of the transmission of rheumatism.

*Season.* In this country chorea shows a most remarkable tendency to make its appearance at certain seasons of the year. The greatest frequency is found in the spring, and the next greatest late in the autumn, or early in the winter. Not only this, but relapses are more apt to occur at these periods. It is worthy of note in this connection that in Philadelphia at least, the seasons above referred to are the ones during which the greatest strain is placed on children by their school-work. It is more than likely, however, that both season and school-work have important parts in the etiology of the disease. Morris Lewis has shown that the occurrence of the disease has an undoubted relation to storm centres.

The poor and underfed are certainly more frequent victims of chorea than are those better provided for. The disease is likewise more frequently observed among those subjected to bad air and overcrowding.

Malaria has been assigned as a cause by Starr. He argues that chorea and malaria are both rare in the negro race; both diseases show a tendency to recurrence in the autumn and spring; and both find an efficient remedy in arsenic, and, as has been more recently suggested, in quinine.

There has been considerable discussion as to the relation between rheumatism and chorea. Some authorities go to the extent of claiming that the former disease acts as a remarkable predisposing, if not an actual immediate cause of the latter. Different observers present statistics exhibiting the greatest variance. Germain See claims to have obtained a rheumatic history in just one-half of his cases; while Steiner finds a rheumatic history in but four out of over two hundred cases of chorea. The former authority included as rheumatic all cases in which there was a parental or family history of that disease, and all cases in which cardiac disturbances were present. Investigations conducted with a large number of cases gave a rheumatic history in about 25 per cent., including in these all presenting a family history of rheumatism, as well as those giving a direct history. Sight must not be lost of the fact that rheumatism in children presents very different picture from that in the adult, its symptoms in the former oftentimes being of the mildest and most evanescent character. It may even happen that the articular complications are slight, and the disease expends its entire force upon the endocardium. The relation as to time between the rheumatism and the chorea is not at all constant. In a few cases, one disease follows the other so closely as to leave the relation of cause and effect almost incontestable. In other cases, we find simply a pre-existence of the former disease. In occasional instances the two affections are coincident. My own observations show apparently that a rheumatic history is obtainable in neurotic constitutions in about the same proportion as it is to be found in choreic patients. This has led me to the conclusion that chorea and rheumatism do not bear an etiological relation, but rather that both conditions are liable to occur in constitutions bearing certain peculiarities.

Sometimes, though rarely, chorea follows the infectious fevers, as scarlatina, measles and whooping cough.

*Pregnancy* appears to be an occasional cause of chorea. It practically never occurs prior to the second month or after the fifth. When once it appears, it is very obstinate to treatment. Patients older than twenty-five years are never thus affected.

Of exciting causes, *fright* and other depressing emotions are the most frequently observed. The duration of time between the reception of the

mental shock and the incidence of the disease is not apt to be long. Sometimes there is practically no interval, the disease coming on at once. In other cases it may be delayed for a week. Sturges has reported some interesting cases bearing on the relation of fright to chorea. Thus a child was frightened by an object which he attempted to pick up, and became choreic in the hand then used. Another child became frightened at a dog jumping at him, and was choreic on that side.

*Excessive mental work or study* most undoubtedly is an active etiological agent in many cases. It must be a prolific cause especially in those whose devotion to duty has already undermined their constitutions and rendered them anæmic. Anæmia is not, however, a pre-requisite to the onset of chorea, for one will frequently meet with cases presenting the highest type of general bodily health.

*Reflex irritation*, as worms, nasal disease, refractive errors, etc., are only occasional causes. Chorea from reflex irritation is apt to present a very different picture from that of the idiopathic disease, the disorderly movements showing a tendency to chronicity and local distribution; the movements, too, are more jerky and angular. The greatest caution must be observed in attributing a given case of chorea to reflex causes, lest pernicious therapeutic activity do more harm than the original disorder. Therapeutic demagogues, as those who practise indiscriminate tenotomies, and their ilk, should receive a deaf ear; many a young girl's life has been wrecked by having her attention directed to her genital organs as the seat of disease early in life. I speak these words to inculcate caution, and not to lead to the opposite extreme.

*Injury* has been assigned as an occasional cause. It is, however, almost impossible to separate the influence of the traumatism *per se* from the depressing emotional effects which must necessarily attend it.

**Symptoms.**—While the mode of onset of chorea is not always the same, still the disease presents a well-defined symptomatic history. Its inception may be either sudden or gradual. In the first case, following the reception of the alleged cause a shorter or a longer time, the child is noticed to begin to twitch, or, more properly speaking, to indulge in restless movements, which rapidly increase in severity and distribution until they are of the most violent character, and affect the entire body. When, on the other hand, the disease is of gradual onset, the usual history is that of a slowly increasing awkwardness or restlessness, for which the child is at times reprimanded. Still another class of cases has the inception of the chorea marked by an apparent loss of power of one or more extremities. When but one member is involved, it is almost always an arm, or part of the same. Sometimes the parents say that one lateral half of the body is losing strength, or they even express fears of an oncoming paralysis. In extreme cases the loss of power may be general, as to amount to an almost complete helplessness. Even in



these so-called paralytic cases, careful investigation shows the characteristic choreic restlessness present to some degree. The apparent paralysis is due more to an indisposition rather than to an inability to move. Sooner or later the choreic movements appear, often being severe in direct proportion to the pre-existing paretic condition.

The choreic movements themselves need a certain amount of description. In a general way, they may be described by one word: "Restlessness." The child is sitting before one, and it will be noticed to lift one arm, and then drop it again, or it will perform supination and pronation; or it shrugs one or both shoulders; twists around in its chair, shuffles its feet; twists the head; makes grimaces of great variety; or these movements may exist together or follow one another with almost kaleidoscopic variety. In mild cases but few of them are to be noted, and these are of slight degree. In the severer ones they are all present, making their victim a pitiable object, reducing her, as they do, to a condition of absolute helplessness. In extreme cases the muscles of deglutition and respiration are affected. Thus nutrition is impaired, while the interference with breathing causes additional discomfort. The respiratory complication may lead even to irregularity in action of the cardiac muscles, though I am inclined to the view that the latter are not infrequently affected *per se*.

In the majority of cases, the choreic movements are either limited to one side of the body, or are much lighter on one side than the other. Usually, the arms are more severely affected than the legs.

The movements cease during sleep. In severe cases they may be of such a degree of intensity as to cause insomnia. This is a serious complication.

Few, if any disorders of sensation are ever noted. Trousseau has observed the frequent presence of numbness and formication. The general trend of observation is to the effect that sensory disorders are absent.

Pain when present is probably of the rheumatic character. Occasionally it may be purely neurotic.

The temperature is generally normal. In severe cases it may occasionally rise as high as 102° F.

Bodily health may or may not suffer as a result of the disorderly movements. Extreme cases have been reported where they have been alleged to have caused rapid emaciation and malnutrition.

The rheumatic and cardiac complications of chorea have ever been a subject of scientific interest and study. Concerning the relations of the disease to rheumatism, I have already spoken when treating of etiology. It is only necessary in this place to refer to subcutaneous rheumatic nodules which are occasionally observed.

The cardiac symptoms of chorea call for more extended notice. It

is acknowledged by all observers that cardiac murmurs are heard in choreic patients with a remarkable degree of frequency. All are not, however, agreed as to the nature of the lesion. The facts from a clinical standpoint are that a soft systolic murmur heard best in the apex, more marked with some beats than others, is often observed in connection with chorea. It is especially apt to be present in the younger patients, those after the age of fifteen almost entirely escaping, and those less than eight being rarely free from it. In the majority of cases, in all so far as my own experience goes, this murmur entirely disappears when the chorea is cured. It gives rise to no symptoms and its presence can only be determined by physical examination. Most authors rightly look upon this murmur as of purely functional nature, and explain its existence by various hypotheses, none of which are sufficiently satisfactory to suit all cases. Others claim for it an inflammatory origin, saying that it is the result of endocarditis, thereby showing it to be of correspondingly serious moment. Osler, for example, has apparently shown that chorea leads to serious organic heart diseases, and gives numerous cases in support of his position. Out of one hundred and ten cases of chorea examined two years after the subsidence of the disease, fifty-four, he says, had undoubted cardiac disease. One of these cases subsequently came under my treatment. My examination satisfied me that the organic heart disease was secondary to kidney disease, for the latter was known to have been present long prior to the onset of the chorea. I have seen one case of fatal heart trouble in which the only assignable cause was a chorea following scarlatina. Kidney complications were absent in this case. In some cases, the murmur is of older date than the chorea. The recognition of such a case is of importance from a prognostic standpoint. We may assume such a state of affairs to be the correct one when the cardiac disease is considerable in degree at an early stage of the chorea, especially when the walls of the heart present consecutive changes, and there is an undoubted history of inflammatory rheumatism to produce such a condition. The choreic heart disturbance is not limited to the murmur above mentioned. In some cases, it may take the form of irregular action, and in still others, simply quickened action. In some few cases there can be no doubt that a purely functional anæmic murmur exists.

Pericarditis is an occasional complication of chorea.

The mental condition of the choreic patient is usually abnormal. In nearly all cases, this abnormality takes the form of remarkable irritability. In some few instances, a more serious condition prevails. Even mania may develop. Delusions may be present, often with wild manic excitement. So severe may the condition become that food has to be administered by the rectum, and stools and urine pass unnoticed.

Speech is not infrequently involved, but usually by reason of exten-

sion of the choreic movements to the muscles of the tongue, or indisposition on the part of the patient to talk. The usual condition is that of indistinct utterance, though it may amount to absolute speechlessness.

The faradic irritability of the affected parts is somewhat increased.

**Pathology.**—The pathology of chorea has been extensively discussed without arriving at a conclusion satisfactory to all. There can be no doubt that there is an instability of motor nerve-cells in the brain, although one author (H. C. Wood) pleads for a possible spinal origin. The cause of the cellular instability is, however, a matter of great doubt. When all facts are considered, I believe that the theory propounded by Sturges, that the instability of the nerve-cells is purely functional in character, is best adapted to explaining the majority of cases. In favor of this view we find the predominance of the disease in females, its dependence upon neurotic inheritance, and its not infrequent occurrence from emotional influences and reflex causes, and its onset during the developmental period of life.

Koch has argued for a microbic origin. He believes in a choreic virus, with the primary affection in the nervous system, the endocardial and articular troubles being purely secondary. Starr, while not accepting this view, gives as points in its favor that tetanus is an infectious disease; that tuberculosis can result from injury, and rheumatism from exposure to wet and cold. He thinks, therefore, it is in perfect accord with these facts to say that when the nervous system is the *locus minoris resistentiæ* chorea may follow a fright and yet be due to a specific micro-organism.

Dana believes chorea to be dependent upon changes in the gray matter of the cortex and its meninges, the pyramidal tract, lenticular nuclei and the spinal cord. The lesion he thinks is an intense hyperæmia with dilatation of the vessels, small hæmorrhages, and spots of softening; infiltration of the perivascular spaces with round cells and swelling and proliferation of the intima of the small arteries. The cause is either micro-organisms or endocarditis. One must readily see how unsatisfactory this pathological explanation is, as it throws but little light upon the disease.

Kirkes is the author of a theory that has always had great weight with the mass of the profession, a theory to the effect that chorea is dependent upon minute emboli affecting the capillaries of the corpora striata. In favor of this are the facts that such lesions have been found in a few fatal cases, and that choreic movements in lower animals have been produced by experimental capillary embolism of the corpora striata.

**Diagnosis.**—It seems to me impossible to confound chorea with any other disease, although some few cases of disseminated sclerosis and Friedreich's ataxia may present a superficial resemblance. This subject



will be found fully discussed in the chapters devoted to these diseases. As to confusing the ordinary chorea of childhood with the other so-called varieties of chorea, I think there should be no difficulty after a perusal of their characteristics. Occasionally, there may be a liability to confuse a restlessness peculiar to the individual with chorea. Then too, those cases of chorea beginning with paralytic symptoms may give rise to error. The most remarkable illustration of this I ever saw was that of a boy who was struck on the arm, and shortly afterwards was noticed not to use that member as before. Fortunately for the sake of diagnostic accuracy, the choreic movements followed the paralysis within twenty-four hours. Blepharospasm and habit twitchings of some of the facial muscles must be distinguished from chorea.

**Duration.**—Chorea is usually said by authors to be a disease of self-limited duration, running its course in from six to eight weeks. This I think is a mistake. The majority of cases of this disease will be found of greater duration. I do not regard this assertion on my part as evidence of inefficiency of the treatment employed, for I find that very many cases have lasted fully two or three months before coming under observation. The acute cases with violent movements are perhaps of shorter duration than the lighter ones, probably because they receive closer attention. The course of the disease may be lengthened to several years.

**Recurrence.**—Chorea shows a remarkable tendency to recur. Generally, this tendency is manifested at certain seasons of the year. It is not uncommon to find patients who have had four attacks, and I have known one instance where the number went up into the "teens." The interval between the attacks varies greatly. Sometimes it is so short as to give the impression that the return is really an exacerbation of the original trouble. When the disease has remained away for eighteen months it is pretty safe to say that it will not occur.

**Prognosis.**—Chorea usually ends in complete recovery. Some of the adult cases may run on for years, uncured. Fatal results are occasionally met with. The mortality percentage of the British Medical Association's Collective Investigation report, namely 2 per cent., I consider altogether too great. From one-half to three-quarters of one per cent. will probably be nearer the truth. I have seen but two fatal cases, both in consultation, and these out of certainly over three hundred cases of the disease.

**Treatment.**—Rest is a highly important measure. If possible the patient should be put to bed. Unfortunately, the general practice is, when the disease is mild, to order gymnastics, out-door sports, etc. Fresh air is of course a valuable adjuvant, but when combined with violent muscular exercise, its propriety is doubtful. It will nearly always be found a good measure to order rest for a period of a week or ten days in the first stages of each case. To lessen the monotony, the little one may be permitted to have its toys in bed.

It is needless to say that the child should be taken from school, and all study positively interdicted.

The diet should be of the most nourishing character. Goodhart and Phillips have reported remarkable results from overfeeding alone. In cases giving evidence of malnutrition, cod-liver oil is of unquestionable value. It is even wise, in my opinion, to administer it as a routine measure in this disease. Fatty food is a great aid in the treatment of all neurotic conditions. Children, as a rule, bear the cod-liver oil remarkably well. The character and quantity of the food and the frequency of feeding must, of course, be prescribed according to the age and general condition of the patient.

Moral management of these cases is very important. Children are great observers, and may be unfavorably affected by injudicious behavior of attendants. Exhibitions of parental worry must be avoided. The medical attendant must ever be cheerful and hopeful in their presence. Perhaps the best device is one which will lead the little patient to look hopefully forward to some pleasant event that will occur when she is well.

In very severe cases, when the movements are violent, it is necessary to take measures to prevent self-injury. Confining the limbs is bad practice. It is better to simply pad the sides of the bed. In these severe cases it is remarkable to notice the improvement that will often follow the absolute rest in bed. In a week or ten days such patients will often quiet down remarkably. Relapses may readily occur, however, from too early getting up.

Sleep is undoubtedly the best form of rest. When, therefore, there is any prospect of its being disturbed, it must be closely looked after. While the general rule is to secure sleep at all hazards, this must not be done recklessly. Hypnotics should never be employed excepting as a last resort. Usually a warm bath, or a hot sponge bath in the evening, will be amply sufficient to secure a good night's rest. In certain rare instances—in cases which will become less and less frequent as one's experience increases—hypnotics will be found necessary. Then chloral combined with bromide of potassium should be given by preference.

Fatal cases of chorea owe their unfortunate termination to profound prostration. This may be avoided by the care bestowed on the patient in the first stage of the disease. When once it has set in, it is to be combatted by the administration of alcohol in full doses. Very frequently the failure in strength is announced by marked dryness of the skin and the refusal to take food. At this stage, a warm wet pack is very often a most valuable adjuvant.

Massage is a useful general measure in subacute and chronic cases. It secures all the beneficial effects of exercise without exposing the patient to the effects of tire.

As to the remedies for chorea, there are none in which I have as much confidence as I have in *agaricine*. This remedy I have been in the habit of using in the second decimal trituration, administering a one-grain tablet of the same every two hours, or, in extreme cases, every hour. I invariably give it when indications call for no other drug. With *agaricus* I have had but little experience, and that unfavorable. Still I have limited its use to cases in which the prominent symptom was twitching of the eyelids, and in which condition *hyoscyamus* has given me decidedly better results. In cases with spinal symptoms it may, however, be borne in mind, as its pathogenesis shows it to be a remedy of value, *e. g.*, spasmodic motions from simple involuntary motion and jerks of single muscles to a dancing of the whole body; trembling of legs and hands; soreness of the spine; aggravations of symptoms during a thunder-storm. The characteristic symptom of *agaricus*, tingling of different parts of the body as if frost-bitten, must not be forgotten.

*Ignatia* should be administered in cases resulting from fright, grief, or other depressing emotional influences.

*Causticum* has been recommended under the same circumstances by Jahr, when *ignatia* proves inefficient. Its special sphere is in cases in which parietic conditions are strongly marked. The right side of the body is especially apt to be involved. The movements are severe; the tongue is affected, so that speech is indistinct, and words are jerked out. *Causticum*, being a good anti-rheumatic remedy, is all the more indicated when a history of that disease is present.

*Ferrum redactum*, as suggested by Hughes, I believe to be a necessary remedy in cases characterized by marked anæmia or chlorosis. The dose is one grain, three times daily after meals.

*Actea racemosa* is indicated both etiologically and symptomatically in chorea. By reason of the former, it is valuable in rheumatic cases, and also in those occurring in girls at the age of puberty. Myalgic pains are apt to be present. The association of the disease with uterine symptoms only serves to indicate the drug more strongly. Reflex neuralgias are prominent symptoms. The choreic movements are worse on the left side. If menstruation has already appeared the movements are apt to be aggravated at such times. Sleeplessness, depression of spirits, and other evidence of mental derangement afford additional indications for *actea racemosa*.

*Pulsatilla*, like the remedy last named, is adapted to cases occurring in girls at the age of puberty. The peculiar *pulsatilla* mental condition is the leading indication of the drug.

*Hyoscyamus*, as already suggested, is adapted to cases in which local twitchings are a prominent feature. It may also be useful in severe cases, even in those in which mania becomes a prominent symptom. Prostration is great.



*Stramonium* is indicated in cases in which the movements are both general and violent. The cerebral condition is the indicating one. The child awakes from sleep screaming, and laughs without reason.

*Veratrum viride* has cured a number of very severe cases when prescribed empirically, as reported in Hale's *New Remedies*. It should be especially useful in cases characterized by rapid pulse, and congestion of the brain.

*Cina* and *spigelia* are said to be adapted to cases arising from the irritation of worms.

*Nux vomica* has cured cases which have been overdrugged, or in which gastric symptoms were prominent features.

*Iodine* is suggested by Hughes in cases presenting a tubercular history. The homœopathicity of this drug to chorea is shown by the fact that twitchings are prominent in the symptomatology of iodism. Jousset reports excellent results from iodine in the graver forms of the disease.

*Zincum* is indicated when the general health suffers much, and fright has been the cause of the disorder. The feet are more markedly affected than usual.

*Zizia* has been recommended where the choreic movements persist during sleep, a rare condition of affairs.

The spider poisons have been strongly advocated as choreic remedies. *Tarentula* especially in cases in which the movements affect the right arm and right leg. Jousset looks upon this as our most efficient remedy in chorea.

*Mygale* was for a long time my favorite remedy. While it did good work in a number of cases, still it did not accomplish what agaricine does now. The provings of this drug were made by Dr. J. G. Houard, who is the authority for the following symptoms: "Muscles of the face twitch; mouth and eyes open and close in rapid succession; cannot put the hand to the face, it is arrested midway and jerked down. Gait unsteady; legs in motion while sitting, and dragged while attempting to walk. Constant motion of the whole body."

*Cuprum* is mentioned by Bæhr as his favorite remedy. He gives it in all cases in which no other remedy is indicated. He claims that chorea rarely lasts more than three or four weeks under its administration.

Other remedies that may prove useful in individual cases are *phosphorus*, *sulphur*, *calcarea carb.*, *cocculus*, *belladonna*, *strychnia* and *phosphoric acid*.

In the chorea of pregnancy, Hale recommends *ignatia*, *hyoscyamine*, *arseniate of iron*, *cuprum arsenicosum*, *kali ars.* and *agaricus*.

*Arsenic* in the form of Fowler's solution, as used by the old school, will prove of value in anæmic cases. The dosage varies largely according to the individual preference of physicians. One old-school physician

makes the extravagant claim that large doses—fifteen minims three daily—will effect a cure in about a week. The majority, however, recommend that the initial dose be five minims three times daily, and that this be gradually increased until from fifteen to twenty-five minims are taken at one dose. Hale is a very strong advocate of the remedy, advising its administration in doses of one drop three times daily to begin with; then the dose is increased by one drop a day for ten days, and then decreased for another ten days. He expects a result within twenty days. My own experience with Fowler's solution as a routine remedy has been disappointing. Occasionally it brings about a most brilliant result. I have used it in all doses, with the exception of the extravagant ones first mentioned.

*Electricity* may be employed in obstinate cases. I much prefer the method of application proposed by Dana, namely, anodal galvanization of the brain. The positive electrode, well moistened, is placed over the motor area on the side opposite to the one most severely affected by the movements. The negative electrode is then placed in the choreic hand. A mild current is then permitted to pass without interruption for two or three minutes. If both sides are affected, then the sides of the electrodes are reversed, the positive one being still kept to the head, and the negative to the hand. Beard and Rockwell recommend general faradization and central galvanization after their peculiar methods. Erb makes applications to the head, so that the motor zones of the brain are situated directly between the electrodes, *i. e.*, obliquely from the region of the central convolutions to the opposite side of the neck (negative, large electrode) for one-half to one minute on each side with a feeble current, or in the manner recommended by Berger, with a bifurcated electrode, the positive upon both parietal regions, the negative being placed in the hand or upon the back for five to ten minutes.

#### MISCELLANEOUS AFFECTIONS BEARING THE DESIGNATION CHOREA.

The class of affections to be considered in this section have been burdened with the name chorea, although they apparently bear little or no pathological relations to it. They have been so designated because they are characterized by certain abnormal movements, supposed at one time to be of choreiform nature.

(a) **Electrical Chorea.**—This is a very rare affection, seen almost exclusively in Lombardy and neighboring parts of Italy. It is characterized by spasmodic movements which consist of sharp quick muscular contractions similar to those produced by electrical excitation with the interrupted galvanic current. These movements commence in one extremity, usually the arm, spread to the corresponding leg, and next invade the other side of the body. They increase gradually in severity.

Finally the affected limbs become weak, and their muscles atrophy. General paralysis finally supervenes. Then the patient dies. But little is known concerning this strange affection. Pathological investigations have been very unsatisfactory. Its causes are also unknown. Fright and malaria have been assigned as causes. It affects both sexes and all ages. It has also been called "*Dubini's disease*."

(b) **Chorea Major.**—This affection has likewise no clinical or pathological relation to the ordinary chorea of childhood. Inasmuch as the common name applied to chorea by the laity, St. Vitus's dance, originated in this affection, chorea major, it has been customary for authors to consider it in this connection. Chorea major is really a form of religious hysteria, characterized by wild dances and great excitement. For the cure of this, it was customary in the middle ages to make pilgrimages, especially to the shrine of St. Vitus. Epidemics of this character are now seldom observed, although they have occasionally occurred in this century, even in the United States.

(c) **Huntington's Chorea; Hereditary Chorea.**—This affection was first systematically described by Huntington, a Long Island physician, in 1872. It had been previously described in 1841 by Waters, of Franklin, N. Y., in a letter to Duglison. The most remarkable feature in the etiology of Huntington's chorea is heredity. It has been known to affect four generations, and as many as nineteen subjects in a single family. If one generation escapes, the line of heredity is destroyed. It does not begin until adult life is reached, generally between the ages of thirty and fifty. The exciting causes of the ordinary type of chorea seem to be productive of this disease at times, though more frequently than otherwise, heredity remains the sole etiological factor. The choreiform movements begin gradually in the upper half of the body, and slowly increase in distribution and intensity. They resemble in character the movements of the chorea of childhood. The eyeballs are often affected, and the mental condition is very frequently considerably impaired. The disease runs a very chronic course of from ten to twenty years. It is essentially incurable. Cases die apparently by reason of the associated mental degeneration. The pathology of the affection is unknown.

(d) **Senile Chorea.**—Choreic movements sometimes attack people of advanced life, even up to the age of eighty years. Cases of this character are very obstinate to treatment, and generally continue until death takes place from other causes. Depressing emotions and the neurotic constitution seem to play the most important part in their causation. The movements are especially liable to affect the upper part of the body, the involvement of the face and tongue being so severe as to interfere seriously with articulation. The presence of strangers or unusual excitement intensifies the movements. The patient is quiet during sleep.



The mind remains unaffected. The general health is not impaired by the disorderly movements. Some few cases recover.

(e) **Habit Spasm ; Habit Chorea.**—This affection consists of definite spasmodic movements, these varying according to the case. Thus in some there is blinking of the eyes ; in others, twitching of the corners of the mouth, twisting the head, shrugging the shoulders, etc. They may come on at any time of life, but occur principally in children. Females are more subject to them than males. Causes tending to constitutional depreciation often act as excitants of habit chorea ; thus we find such patients giving a history of preceding acute diseases. Emotional influences and agencies depressing to the nervous system as excessive mental application are likewise causes. Cases have come on after an attack of meningitis. The frequency of the spasmodic movements presents great variety. They may occur all the way from every few seconds to a much longer time. No portion of the body is exempt from the movements. Even the respiratory muscles may take on a spasm. This is evidenced by sudden crowing inspiratory sounds, sniffing, coughing, etc. The spasmodic movements vary in severity from very strange causes. Sometimes, the presence of strangers is sufficient to drive them away entirely. Drawing attention to them very frequently intensifies them. The prognosis depends largely upon the age of the patient and the duration of the disease. Cases in children nearly always recover after a time, although they are usually very obstinate. Adult cases are not so promising, while those occurring in advanced life are practically incurable. The *treatment* includes attention to the general health as the first essential measure. The family and attendants should take no notice of the trouble. In some cases it may be wise to send the patient away from home for a time. As to remedies, the reader is referred to those mentioned under chorea.

(f) **Tic Convulsif.**—Tic convulsif is characterized by paroxysms consisting of sharp electric-like contractions of single muscles or groups of muscles. Following these paroxysms there is a period of rest. The movements are generally limited to the distribution of a single nerve or nerve branch.

(g) **Gilles de la Tourette's Disease.**—This is a tic convulsif, to which are added certain characteristic disturbances of speech and mind. The patient gives forth explosive utterances or cries ; or he repeats or mimics words he hears. To this latter the term *echolalia* has been applied. In other cases, the patient at the time of the spasmodic movements utters obscene or profane words. This is called *coprolalia*. Sometimes he mimics gestures made before him. This is known as *echokinesis*. Again, he may speak out the thought he may happen to have in mind (*tic de pensée*). Gilles de la Tourette's disease generally affects children between the ages of six and sixteen. It is an exceedingly chronic disorder.

(h) **Saltatoric Spasm.**—Saltatoric spasm was described by Bamberger. It is characterized by clonic spasm of the leg muscles, coming on as soon as the patient attempts to stand. These movements may be but temporary, or they may be so persistent as to be a source of true disability.

(i) **Myriachit; Latah.**—Myriachit is synonymous with a peculiar nervous affection observed in certain communities in Maine, Canada, Java, Siberia and Kamschatka, in each bearing a peculiar provincial designation. Thus we hear of the “Jumping Frenchmen” of Maine. The disease is characterized by sudden movements from the slightest touch or noise. Sometimes the patient has an irresistible tendency to imitate acts seen, or obey the most absurd commands given him. Thus these jumpers, if ordered to throw a knife, will throw it; or, at command, will jump overboard. The impulse to do so is irresistible.

(j) **Imperative Movements, Head-Nodding, etc.**—Idiotic and imbecile children are often the subjects of imperative movements due to dominant conceptions and insistent ideas. These may consist of almost any kind of movements or combination of movements.

*Head-nodding* is a condition first systematically described by Hadden. The movements consist of nodding or lateral movements of the head, “either singly or associated with one another, or with movements of rotation. Further, these movements of the head may be almost constant, or may occur, more especially, during efforts at fixation or during excitement, always ceasing during sleep or when lying down. In most cases there is nystagmus of one or both eyes, vertical, horizontal or rotary, often occurring simultaneously with the head movements, but sometimes preceding or following them. The nystagmus is much more rapid than the head movements, and has an independent rhythm; it is aggravated by attempts at fixation or by forcibly restraining the head, and may be induced, when previously absent, by these means.” (Hadden.)

*Head-banging*, consisting of a condition in which the child turns over on its face at night and bangs its head into the pillow, has been described by Gee. It seems to be a rare condition.

## PARALYSIS AGITANS.

Paralysis agitans is a chronic nervous disorder of uncertain pathological nature, characterized by tremor *in association with* more or less general rigidity, muscular weakness, and peculiar gait and posture. It was first described in 1817 by a Mr. Parkinson, hence its synonym, Parkinson's disease. It is commonly spoken of by the laity as shaking palsy.

**Etiology.**—But little is positively known concerning the etiology of paralysis agitans. Strictly speaking, it is not a disease of old age; and yet its characteristic features apparently indicate that it results from a premature senility. The majority of cases commence between the fiftieth and sixtieth years of life. It very seldom indeed occurs prior to the age of forty. Cases have been described as occurring in youth, but these are so rare as to be regarded as clinical curiosities. Males are more frequently the subjects of the disease than females, probably because they are more liable to exposures of various kinds and to greater responsibilities and anxieties, these latter being well recognized as having an active etiological influence in certain cases. Some few instances have followed traumatism. Alcohol and syphilis certainly, and sexual excesses probably, have no part in the production of paralysis agitans.

**Symptoms.**—Paralysis agitans is in the vast majority of cases of insidious onset. Some few cases, however, have been reported in which the symptoms have made their appearance with almost the suddenness of an apoplectic seizure. Such an onset usually follows a great shock or other powerful exciting cause. The first symptom observed is a tremor of mild degree, gradually increasing in severity over a term of years. Almost always this tremor begins in one or the other hand. It then spreads to the legs on the corresponding side; then crossing to the other side of the body, involves the arm and leg in order. It may exceptionally invade both hands and extend to the legs, or it may involve the arm of one side and the leg of the other, or again both legs only may be affected. In the early stages of the disease, the tremor is observed only when the affected part is at rest. Attempts at voluntary motion control the movements completely. Later, however, motion exerts no influence whatever over them; in fact, it may have the reverse effect, that of greatly aggravating them. The tremulous movements are decidedly rhythmical, ranging from 3.7 to 8 per second. As involving the upper extremity, they may be described as follows: The hand is held in the writing position; the tremulous movements in the fingers consisting of metacarpo-phalangeal flexion and extension bearing similarity to the



movements employed in spinning wool (Charcot) or rolling a pill. There are also rhythmical flexion and extension at the wrist, and supination and pronation at the forearm. The arm is flexed at the elbow. The tremor ceases entirely during sleep.

Muscular weakness is a constant symptom, though not a prominent one. Occasionally it may exist to a marked degree, under which circumstances the tremor is proportionately severe. Still more rarely, the weakness may be great and the tremor entirely absent. The conclusion that a slowly progressive hemiplegia is at the bottom of the trouble seems almost irresistible. The muscular weakness in paralysis agitans never amounts to complete loss of power.

The rigidity and the peculiar attitudes of the patient are as characteristic of the disease as is the tremor. The muscles everywhere are rigid, this condition being especially marked in the flexors. The back is bowed; the head is flexed on the chest. The arms are held flexed at the elbow and slightly away from the body. The hands are in the writing position, and drawn slightly towards the ulnar side of the forearm. The legs likewise show involvement. With the patient standing there is slight flexion at both hips and knees. The muscular rigidities and contractures are the cause of the characteristic gait. The patient moves as if he was soldered together, so to speak, at his joints. In starting to move, he manifests considerable difficulty, but having once begun, he proceeds fairly well. He may even show a tendency to run, taking quick, short, shuffling steps. The body being bent forward with this gait, has led to its being described "as if the patient was pursuing his own centre of gravity." Charcot has called this phenomenon in walking, "propulsion." A very slight push will cause the patient to pitch forward. A slight pull backward will cause him to fall in that direction, this being called "retropulsion." In some few cases a lateral pull will produce "lateropulsion." Charcot believes these phenomena partake of the nature of forced movements, though Strümpell with apparently good reason thinks that they arise from the displacement of the patient's centre of gravity by his posture. The muscles of the face exhibit their involvement by their immobile expression. The trunk seems to be more affected than the extremities. It is said that the muscles of the eyes are rarely implicated.

The speech exhibits several abnormalities. The most constant is a peculiar monotonous, expressionless voice. Another, first described by Buzzard, consists of a shrill piping tone comparable with that assumed by actors in their stage impersonations of aged men. And still another is the vocal counterpart of the gait, if the reader will permit the expression. The patient finds considerable difficulty in starting his speech, but having once begun proceeds with comparative fluency.

The tendon reflexes are usually normal; occasionally they may be

exaggerated, though never to the extent found in the organic nervous diseases in which such a condition is observed.

The sensory functions are unimpaired. Osler claims, however, that there are some alterations in the temperature sense. The patient frequently complains of subjective sensations of heat, which may be accompanied by an actual increase in the surface temperature. The internal temperature of the body is normal in these cases.

**Pathology.**—There is absolutely nothing reliably known respecting the anatomical lesions of paralysis agitans. The most rational view of its nature is that which attributes it to a premature senility. Though many autopsies and investigations have been made, no constant results have been found.

**Diagnosis.**—It is the usual claim of the books that paralysis agitans is most frequently confounded with disseminated sclerosis of the brain and spinal cord. The symptomatologies of the two diseases present great distinctions. The tremor of disseminated sclerosis is of much wider amplitude than that of paralysis agitans; in fact, it partakes largely of the nature of inco-ordination; it is not rhythmical, and occurs only during voluntary muscular movements. The head is frequently involved. Charcot has laid great stress on the non-participation of the head in the tremor of paralysis agitans. Gowers, Strümpell and others have shown, however, that the head is exceptionally involved; the latter further adds that in such cases the muscles about the mouth and chin are principally at fault. Disseminated sclerosis lacks the characteristic rigidity, is often accompanied by nystagmus, and is more apt to attack young adults. The tendon reflexes are usually greatly exaggerated. Pupillary abnormalities are not infrequent. The mind may be affected. The speech has been described as "scanning."

Error may arise in failing to recognize those exceptional cases of paralysis agitans in which tremor is absent, and rigidity and weakness are marked features. The peculiar attitude of the body, the normal condition of the deep reflexes, and the absence of any rheumatic disease, should make the diagnosis easy.

Senile tremor is sometimes mistaken for paralysis agitans. The movements are, however, of much finer range than in that disease. They are aggravated during attempts at voluntary motion. Senile tremor usually begins in the head and neck, the hands and arms being subsequently involved. It is not to be observed in the hands when they are at rest.

**Prognosis.**—No disease offers a more unfavorable outlook, so far as benefit from treatment is concerned, than does paralysis agitans. It may be looked upon as absolutely incurable. Of all old-school authors, Landon Carter Gray is the only one who offers any hope whatever for the sufferer from this disease. By a combination of medicinal and gen-

eral therapeutic measures, he positively asserts that a majority of cases can be so ameliorated as to enable them to live comfortable lives for years. Some few cases have been reported as greatly benefited; but these are so exceptional as throw a doubt on their true nature. It rarely destroys life. Usually the patient lives on for years, twenty or thirty perhaps, finally dying of some intercurrent disorder.

**Treatment.**—Warm baths and massage comprise the general measures that do the most good. They must, of course, be persevered in for years. Rest from fatigue is necessary. Electricity, a weak galvanic current through the head, has been recommended by Eulenburg.

Beard and Rockwell claim some curative results from general faradization and central galvanization, though they think that the best results will be obtained by galvanization of the spine, sympathetic nerves and the brain. Gray's method consists in the application of a large, flat electrode to the nape of the neck, and another, somewhat smaller, to the lumbar spine. A current of from three to five milliamperes should be passed for five minutes. Later, this may be increased to fifteen or twenty milliamperes, and the sitting to fifteen or twenty minutes. The seances should be given at least three times a week.

Palliative medication in the shape of the administration of drugs that will do away with the tremor, is about all that is offered by allopathic authorities. One author praises atropia in doses of one-sixtieth of a grain daily as efficient. Seguin pins his faith on pure crystallized hyoscyamia (Merck's). One one-hundredth of a grain is the normal dose by the mouth. If this drug be given in doses of sufficient strength to produce its physiological effect, such as dryness of the mouth, etc., it will stop the tremor in most cases. Experience will teach, however, that some patients will prefer to suffer from the objective symptom tremor, than to be annoyed with the subjective discomforts of a dry mouth and throat.

Homœopathic literature shows but few cures, and these by *plumbum*, *mercurius*, and *tarentula*. The latter drug was prescribed, with good effect, on purely symptomatic indications. The other two were given more particularly because of the prominence of tremor in their symptomatologies.

Hale speaks favorably of *bromide of potassium*, *calabar bean*, *cannabis indica*, and *oleum jecoris aselli*. From the context one is forced to believe that he confuses a number of pathological conditions under the head of paralysis agitans. Still the remedies he mentions are all such as one would judge *a priori* from their pathogenesis to be useful in cases presenting tremor as a prominent symptom.

*Agaricus* cured for Hughes a case of tremor of long standing, in an old man. It had the peculiar feature of twitchings of the arms, ceasing when he used them at his work of shoemaking. Drop doses of the tincture were administered.



*Baryta, bufo, ergot, gelsemium, hyoscyamus, rhus, tabacum* and *zinc bromide* may also be studied.

### TREMOR.

**Definition.**—Tremor consists of involuntary oscillatory movements, which are produced by more or less rhythmical alternate contractions and relaxations of antagonistic muscles. It is to be distinguished from fibrillary contractions, a condition characterized by alternate contractions and relaxations of individual bundles of muscular fibres, manifested, not by gross movements of the parts to which the affected muscles are attached, but by wavy subcutaneous oscillations of the fibres involved. Nor should tremor be confounded with muscular twitchings, the muscular contractions in the latter being more decided and less persistent. Clonic movements arising from tension on tendons in cases in which the deep reflexes are greatly in excess, have been erroneously denominated tremor by some; many of the so-called tremors in spinal and cerebral syphilis and after hemiplegia are undoubtedly of this class.

There are certain characteristics in every case of tremor that should be carefully noted. They are: (1) The amplitude of the tremor, whether the movements are fine or coarse. (2) The rapidity and regularity of the movements. (3) The regularity of the tremor and the influence exerted upon it by certain external agencies, such as rest, voluntary movements, sleep, massage, etc. (4) Its distribution, for it may be limited to single parts or limbs, or to one lateral half of the body; or again it may be of universal distribution.

It has been customary for years, in treating of the subject of tremor, to divide cases into two great classes, one, *tremor coactus*, in which the movements persist during repose; and the other, *tremor a debilitate*, in which they are observed only during efforts at voluntary movement. This classification is, in my opinion, not as valuable clinically as we have been led to believe, for many of the cases of tremor assigned to the former class have the characteristics of the latter, and *vice versa*. Regarding the different influences which ameliorate tremulous movements, there is one that I do not find mentioned at all in any literature at my disposal. I refer to massage. Several cases of the most aggravated character have shown a cessation of the tremor for several seconds following manipulation of the muscles involved, or pressure upon certain portions of the trembling extremity. None of these cases was hysterical.

Tremors persisting during rest are typified in paralysis agitans; in fact this disease is almost the only one in which the movements cease during voluntary movements. Tremors arising from senility, toxic influences, asthenia, hysteria, disseminated sclerosis, and organic disease of the brain, are mostly observed during attempts at voluntary motion. Cases of paroxysmal and epileptic tremor cannot be assigned to either

class. They are distinguished from all others in that they occur under such conditions as to make the execution of voluntary movements impossible.

I shall treat of the various forms of tremors under the following heads:

- (1) Simple tremor, including the so-called essential tremor, and cases arising from hereditary transmission.
- (2) Senile tremor.
- (3) Asthenic tremor.
- (4) Hysterical tremor.
- (5) Toxic tremor, which may arise from exposure to the following poisons:
  - (a) Lead.
  - (b) Tobacco.
  - (c) Mercury.
  - (d) Alcohol.
  - (e) Chloral.
  - (f) Opium.
  - (g) Tea, coffee, etc.
  - (h) Uræmia.
- (6) Tremor symptomatic of or associated with the following named diseases:
  - (a) Exophthalmic goitre.
  - (b) Writers' cramp.
  - (c) Epilepsy.
  - (d) General paralysis of the insane.
  - (e) Disseminated sclerosis.
  - (f) Paralysis agitans.
  - (g) Tumor of the brain.
- (7) Post-paralytic tremor.
- (8) Paroxysmal tremor.

In the above classification no mention has been made of acute or transient tremors, such as those arising in the apparently healthy as a result of physical or mental strain, exposure to cold, etc. The nature of tremors arising from these causes is readily recognized by the merest tyro, and their consideration is unnecessary because they are of so short duration as to rarely, if ever, be brought to the attention of the physician.

*Simple tremor* includes all those cases in which the tremor occurs without apparent cause, or is unassociated with other evidence of ill-health. In many instances it has shown a remarkable tendency to occur in families, so much so in fact as to lead some authors to establish a separate class called hereditary tremor. In all these cases the tremulous movements are very fine and generally regular as to their amplitude and rhythm. They are for a time under the control of the will. While

intensified during voluntary movements, they do not interfere in the least with the execution of the same. The patient can perform such complex movements as writing without difficulty. Though not necessarily so, the tremor is limited to the hands and head. It affects mostly the young and middle-aged. It is very intractable to treatment. Once it has thoroughly established itself, it persists throughout life. Some tremors occurring in children born of nervous mothers disappear spontaneously or under treatment after a few years.

The hereditary tendency to tremor displayed by some families is truly remarkable. Thus, in a case reported by Dr. Samuel West, the patient's grandmother and mother had been affected. He was one of ten children, all of whom suffered from tremor varying in degree, and the affliction has been reproduced in his own six children.

*Senile tremor* appears in people of advanced years and in the prematurely aged. It presents points of similarity to each of several varieties of tremor. It is a fine tremor, aggravated by movement, beginning usually in the head and neck, the arms and hands being attacked after the trouble has lasted some time. Clinically, it is especially apt to be confounded with paralysis agitans. In the latter the tremor is present when the hands are at rest, the movements usually begin in the hands, while the head and neck are generally free, and the tremulous parts are affected with a peculiar rigidity.

*Asthenic tremor* has all the characteristics of the simple variety, but differs from it in having a well-defined relation to certain asthenic conditions. It is especially liable to occur after exhausting diseases, as the specific fevers, in cases of nervous exhaustion, and after hæmorrhages and sexual excesses. Many of the cases of tremor in young males arise from the latter cause. The tremulous movements occurring during the course of diseases other than of the nervous system are of the asthenic variety. They are significant only of the extreme debility present.

*Hysterical tremors* are exceedingly irregular in every respect. They may be fine or coarse, rapid or slow, mild or severe, and these in the one patient. Their distribution is as varied as their character. They may be hemiplegic in type or they may invade the entire body, or be limited to but one limb or to one group of muscles. The movements are intensified during the execution of voluntary movements, but, as a rule, the aggravation does not begin immediately after the movements are commenced, usually not asserting itself until muscular action has been maintained for some little time. Hysterical tremor is almost always increased when attention is directed to it, and ceases when the mind is diverted. It may in some cases be associated with paralyses and contractures. When the limbs are the seat of contracture, the tremor is excited by any attempt to move them passively or even by the manipulation necessary during a medical examination. A long duration (*e. g.*,



many months) does not contraindicate the hysterical nature of a given tremor.

As already stated, chronic tremors may be produced by exposure to certain poisons, such as lead, mercury, tobacco, alcohol, tea, coffee, chloral, opium, etc. There is little to say that is diagnostic concerning tremors arising from the influence of these poisons. They are all fine tremors, usually intensified during voluntary movements. Saturnine tremor is commonly limited to the face and hands. Occasionally, it is very acute and violent. It is aggravated after prolonged exercise, or in the latter part of the day, when the muscular system is exhausted. The diagnosis depends upon a history of exposure to lead poisoning, the previous existence of lead colic, blue line on the gums, anæmia, wrist drop, etc.

Tremor is of far more frequent occurrence in cases of poisoning by mercury than by the metal above mentioned. It usually begins in the face and tongue, and is always intensified by any motion. In its incipient stages, it occurs only during voluntary movements, but later it becomes constant, in which case it presents a remarkable similarity to paralysis agitans. Articulation is very much interfered with. In some cases the tremor is of sudden onset. The diagnosis rests upon the known exposure to fumes of mercury, and upon the associated symptoms. A very interesting as well as rather unusual case of mercurial tremor has been reported by Letulle. A man æt. 39 years, had been exposed to the fumes of mercury for many years. In his first attack of tremors, the movements were worse in the morning, and much relieved between the hours of 4 and 5 p.m., and again very marked in the evening. One day the tremors suddenly became very violent, so much so in fact that the man was thrown down by them, and convulsed, so that he could not walk. When perfectly at rest in bed the movements were but slight; but they were very much intensified by voluntary effort. Motions were extensive, rapid and arrhythmical, and worse on the left side. An anæsthetic area was discovered on the back of the right forearm and hand. Vision had become impaired. Constricting the limb lessened the tremor. Finally the tremor was stopped permanently by ligaturing the limb, and passes with a magnet. The author called it a case of mercurial hysteria.

Acute and chronic alcoholism are almost always attended by tremor of some kind, noticeable usually in the arms, face and tongue, often associated with local muscular twitchings. In severe cases the latter amount almost to shock-like muscular contractions. The alcoholic tremor is especially apt to be worse in the morning, and is promptly relieved by a drink of whiskey. The restlessness of alcoholic subjects makes the tremor very prominent.

Tremor is not a common symptom of the chloral habit. When it occurs as a result of addiction to chloral or opium, it resembles in every way that arising from alcohol.

The tremor caused by excessive use of tobacco, coffee, tea, etc., is of very fine character, apparently presenting no distinctions from simple tremor.

Charcot has described a form of tremor closely resembling that of paralysis agitans, due to uræmia.

Tremor is met with as a prominent symptom in quite a variety of diseases. In *exophthalmic goitre* it is rarely absent. It is generally not of a severe grade, usually being mentioned by the patient as a nervousness, and observed by the physician as an exceedingly fine tremulous movement. Still it may be of variable character. Sometimes it is so violent and regular as to resemble that of paralysis agitans. It may be general or confined to the hands alone, or even accompanied by fibrillary contraction of the muscles of the face. This fact must be borne in mind lest a diagnosis of general paralysis be not too hastily made, especially as exophthalmic goitre is not infrequently associated with mental symptoms. The usual mental anomaly, however, is melancholia, or melancholia alternating with periods of considerable excitement. It has been known to be unilateral in distribution, when the exophthalmus or the goitre was one-sided.

Tremor is occasionally met with as the almost exclusive manifestation of *writers' cramp*. It is then most marked in the forefinger when the hand is at rest, and the fingers slightly separated. It is especially apt to be produced by prolonged work and over-fatigue.

Rare cases of *epilepsy* are met with in which the seizures are preceded by a general or localized tremor as a kind of aura, or as in one of my patients, tremor took the place of the convulsions. Gowers holds that "fits" ushered in by tremor are almost always epileptic, and not hysterical.

*General paralysis of the insane* in its earliest stages presents more or less tremor. The movements are especially marked in the tongue and lips, less so in the fingers. Speech becomes hesitating and drawling, with a tendency to slur words. Its relation to the severity of other symptoms is decidedly irregular. In the final stages of the disease, violent tremor accompanies every voluntary movement.

*Disseminated sclerosis* and *paralysis agitans* are, of all diseases, the most constantly associated with tremor; in fact we might call tremor a necessary part of their symptomatology. These diseases have a well-defined clinical course, the consideration of which has been presented in previous articles.

Some cases of *brain tumor* present tremor as a prominent symptom. Usually, it has but little significance. Sometimes, however, pressure of a cerebellar tumor upon the pons varolii or medulla oblongata, or of a tumor of the centrum ovale upon the fibres of the internal capsule causes a persistent tremor. In a case of cerebellar tumor (sarcoma of the dura

mater) under my care, and in which there was marked irritation of the pons, there were repeated paroxysms of tremor, limited to the arm on the side opposite to the lesion. The attacks lasted all the way from a few hours to a day or more.

Following the paralytic seizures, we sometimes have a tremor affecting the paralyzed extremities. This varies in different cases. Sometimes its character is such as to lead to its being called post-paralytic chorea.

*Paroxysmal tremors* have been described by different authors. Suckling reports the case of a laborer, aged 60 years, who for two years had suffered from attack of tremor of the right upper extremity. Attacks were induced by sudden noises or shocks. The movements were limited to the right upper extremity only. They were of great amplitude, and consisted of very rapid flexion and extension of the forearm, which could not be controlled voluntarily or by force. There was no aura preceding the attack, but coincidently with the onset of the tremor, the patient had pain in the upper part of the right thigh. There was no loss of power in the right arm or leg, and no alteration in the reflexes. The attacks ceased abruptly and spontaneously, as the patient said, with a snap. Dr. Suckling expressed the opinion that the tremor would eventually become continuous, and the case end in paralysis agitans.

The convulsive tremor of Hammond, described by him in 1867 (and in his work on *Nervous Diseases*, 1886), as including those cases of non-rhythmical tremor or clonic convulsive seizures which are unattended with loss of consciousness, but which, nevertheless, are paroxysmal in character, in the light of modern neurology requires no consideration. They may all, or nearly all of them, be relegated to various forms of tolerably well understood nervous conditions, without the necessity of manufacturing a new disease.

As to the possibility of simulation of tremor, I believe that it can be done, and think that I have seen one such case, one in which the movements were limited to one arm, and which ceased on the recovery of substantial damages.

### PARAMYOCLONUS MULTIPLEX.

Paramyoclonus multiplex is probably better included under the tremors than elsewhere. It is an exceedingly rare disease, not more than a dozen cases of the same having been reported. It is characterized by paroxysmal clonic movements, symmetrically distributed in muscles, or groups of muscles, of which one extremity has an attachment to the trunk. The muscles of the forearms, wrists and hands, and legs and feet, are not affected. The majority of cases occur in males. The prognosis is favorable, although the disease runs a slow course.



## TETANY.

To give a definition of tetany that will accord with the descriptions of the disease as presented by all prominent authorities is well nigh impossible. In general we may describe it as an affection characterized by tonic spasms, these *always* affecting groups of muscles bilaterally. The spasms may be either continuous or prolonged; and are unattended by loss of consciousness. Tetanilla is generally stated as synonymous with tetany. Althaus describes a spasmodic affection by this name, and looks upon it as entirely separate from tetany.

**Etiology.**—Prominent among the predisposing causes of tetany is age, the vast majority of cases occurring in children or young adults. Among children, it is especially frequent in those ranging from one to five years of age. Males are slightly more predisposed than females. Of the exciting causes, certain influences tending to constitutional deterioration stand forth prominently. Among these diarrhœa takes first rank. Similar exhausting agencies, as lactation, hæmorrhages, and persistent vomiting, are also causes. Pregnancy is credited with occasionally giving rise to the disorder, but in those cases which I have seen reported in journalistic literature, persistent vomiting or albuminuria has been present as a complication. Exposure to cold and the vicissitudes of weather is the next most frequent cause. Indeed the relation between the disease and exposure led some of the earlier observers to regard it as a rheumatic affection. Certain acute and infectious diseases are followed in exceptional cases by tetany. They are smallpox, typhoid fever, inflammatory rheumatism, measles, febricula, pneumonia, and malaria. A very dangerous form of the disease has been observed to occur in association with, and it is believed in consequence of, dilatation of the stomach. It has even been claimed and with good reason, that certain deaths following washing out of the stomach, are from tetany excited by the passage of the stomach tube. A respectable number of cases have been known to follow removal of the thyroid gland. Thus Wöffler reports that out of seventy cases of thyroidectomy collected by him, seven subsequently had tetany. The latter affection usually appeared within ten days after the operation. Tetany has been observed in a number of instances to occur in epidemic form. The authenticity of these seizures has been questioned, the possibility of their being hysterical not having been satisfactorily eliminated.

The frequency of tetany is debatable. The majority of authorities have seen but one or two cases; others, notably J. Lewis Smith, write as if they had had a large experience in its management.

**Pathology and Morbid Anatomy.**—Here we have a question admitting of considerable discussion. Owing to the small mortality of the disease and its rarity very few autopsies have been made, and these have furnished practically no information. Speculation as to the origin of the disease is great. One authority says, and his view is to me the most reasonable, that the spasms are the result of peripheral irritations acting upon irritable nervous constitutions. Gowers, on the other hand, makes the claim that the neuropathic constitution exerts no influence in the causation of the disease. Another writer says it is the result of the dehydration of the tissues by exhausting diseases; Gray and Osler seem to favor the microbic theory of the affection. And so I might occupy pages detailing the many views expressed concerning the pathology of tetany.

**Symptomatology.**—The disease may or may not be ushered in by premonitory symptoms. If these are present, they consist of headache, pain in the back, and numbness and tingling in the extremities. Even sympathetic vomiting may be present. Then the spasms appear. They are *always* symmetrical, and invade the hands and feet first. In mild cases and in those occurring in children with rachitis and from debilitating causes, they may not extend any further. The spasms, as already stated, are of tonic character. The fingers are flexed at the metacarpophalangeal joints, and extended at the phalangeal, while the thumbs are drawn into the palms. The wrists are slightly flexed. If the shoulder joints are involved, the arms are held in rigid adduction. The feet are extended and inverted, and the toes strongly flexed. The knees are rigidly extended. In severe cases, the rigidity extends to the muscles of the trunk and face. Very exceptionally, the jaws are rigid. The angles of the mouth may be drawn, exposing the teeth. It is even possible for the muscles of the eyeballs to become affected, in which case we have strabismus. The muscles of the tongue, pharynx and larynx are seldom affected; yet their implication is not impossible. The duration of these spasms presents the greatest variations, ranging as it does all the way from fifteen minutes to several weeks. They may be continuous or intermittent. They may continue during sleep, though generally mitigated somewhat during that period. They may even begin during sleep. When the contractions are severe they excite pain. The temperature is usually normal, though it may be elevated, rarely, however, to more than 101° F. A very interesting phenomenon observed during the interparoxysmal period is that known as Trousseau's symptom. If pressure be made for a number of seconds on the artery or one of the main nerves of an affected limb, the spasm is re-excited. This is regarded by most authorities as a very important diagnostic symptom. By others it is not looked upon as constant. The electrical excitability of both nerves and muscles is greatly increased to

both faradic and galvanic currents. The mechanical irritability of the muscles is likewise increased; pinching or stroking them serves to bring on a contraction.

**Diagnosis.**—From *tetanus* it is to be distinguished by the absence or late occurrence of trismus, which is one of the earliest phenomena in true tetanus. The previous history of the case is likewise a guide.

From *hysteria* tetany is differentiated by the bilateral distribution of the spasm. In hysteria the contractures are nearly always unilateral.

**Prognosis.**—The prognosis of tetany is nearly always favorable. Cases occurring in association with dilatation of the stomach offer little hope of recovery, nearly all of them dying. Cases occurring after thyroidectomy have a bad prognosis.

**Treatment.**—The treatment of tetany resolves itself into attention to the causative factors, the diarrhœa, or other cause of debility. The patient must be kept as quiet as possible, and not permitted to be disturbed during the quiescent periods. Nourishment must be of the best quality, and preferably liquid, and of course easily digested.

I know of no homœopathic experience with tetany. On theoretical considerations, I would recommend recourse to remedies indicated by the systemic condition and the provocative factors. From this standpoint almost any remedy may be applicable. The symptoms of tetany of themselves suggest *nux vomica* and *secale*.



## HEADACHE.

The frequency with which the physician is consulted for the relief of headache and the fact that head-pains oftentimes constitute the first symptom of serious diseases make it a matter of importance that I shall treat of this subject rather elaborately. The early stages of many of the acute ailments are sometimes preceded for some days by headache; and although these as well as the cephalalgias arising from certain toxæmias are in no sense nervous ailments, still their neurological relations are such as to make their consideration in this place perfectly proper.

In no class of ailments is the question of etiology of more importance than in the one now under study; in fact, I might say, that given the cause of the headache in a given case, its prompt cure becomes a comparatively easy matter. I shall endeavor, therefore, to follow a classification based on etiology.

In the clinical study of any case of headache, there are certain points that must be carefully inquired into. Is the pain constant or paroxysmal? If the former, does it vary much in its intensity? If the latter what is its frequency, and what is the usual duration of the attacks? Is it a pain, or a mere uncomfortable sensation? Do the paroxysms have a regular cycle, never to be departed from? What is the character of the pain, whether throbbing, sharp, dull, etc.? What is its location? What are its associated symptoms? How is it affected by certain actions on the part of the patient, as use of the eyes, change in posture, sneezing and coughing, pressure over the seat of pain, and how by certain extraneous agencies, as weather, light, noise, heat, and cold? As pertaining to associated symptoms, it is especially important to investigate the condition of vision, and the presence or absence of optic neuritis and errors of refraction, to inquire concerning the digestive functions, the presence or absence of nausea and vomiting, and the association of certain nervous disturbances, as numbness and tingling, exhaustion, etc.

The character of the pain offers us but little help in determining its origin; of more importance are the associated symptoms and the modalities that ameliorate or aggravate the ailment.

In presenting a classification of headaches based on their etiology, one must necessarily tread on debatable ground, for certain headaches are variously classified by equally eminent authorities; thus the headaches associated with gastric disorder are regarded by some as toxæmic, and by others as purely reflex; those of contracted kidney as toxæmic (uræmic) and circulatory. The following classification is not offered as one absolutely correct, but merely as an aid to clinical study:

- (1) Headaches symptomatic of organic disease of the brain and its membranes, as :
  - (a) Brain tumor.
  - (b) Brain abscess.
  - (c) Meningitis.
  - (d) Hydrocephalus.
  - (e) Intracranial hæmorrhage.
  - (f) Dural disease.
- (2) Circulatory. Including,
  - (a) Congestive.
  - (b) Anæmic.

Under this head may also be considered certain of the headaches accompanying contracted kidney, when such are not uræmic, but are purely the result of the increased arterial tension, and certain cases arising from traumatism.

- (3) Headaches originating in certain toxæmia, as :
  - (a) Uræmia.
  - (b) Lithæmia.
  - (c) Rheumatism.
  - (d) Septicæmia.
  - (e) Diabetes.
  - (f) Malaria.
  - (g) Syphilis.
  - (h) Gastric disorders with retention and decomposition of food, etc., in the gastro-intestinal tract. Under this head may be included the cephalalgia from constipation.
  - (i) Poisons introduced from without, as alcohol, lead, tobacco, and noxious gases.
- (4) Headaches reflex from disease in adjacent or distant organs, as :
  - (a) Eyes.
  - (b) Nose and throat.
  - (c) Ear.
  - (d) Intestinal tract.
  - (e) Sexual organs.
- (5) Neuropathic, as :
  - (a) Epileptic.
  - (b) Hysterical.
  - (c) Neurasthenic.
  - (d) Hemicrania or migraine.
  - (e) Certain essential headaches, the causes of which have been hitherto undiscoverable.

It is important that the physician do not confound headache with other painful affections of the head, such as are caused by disease of the cranial bones and their investing membrane, carious teeth, rheumatism

of the scalp, and last but by no means least, glaucoma. The mere act of calling attention to osseous and periosteal disease is all sufficient to prevent diagnostic error where these are concerned. It may be borne in mind that in the majority of cases, but by no means in all, the augmentation of the pain on pressure, affords evidence that the disease giving rise to it is extra-cranial.

Decayed teeth have been known to produce severe headache, mental disturbance, and defective vision. Concerning the importance of the teeth in the production of pains other than toothache, I have already spoken when treating of painful affections of the face.

But of all affections glaucoma is one that must be differentiated from headache. A mistake is rendered easy by the comparative rarity of this affection in the experience of the general practitioner, and by its occurrence in constitutions supposed to favor the origin of headaches. The pains may be so located as not to suggest the eyes as their cause. The visual disturbance may be attributed to the same vaso-motor changes that occur in ophthalmic migraine. In glaucoma, there is a concentric diminution in the field of vision, which is more or less permanent; while in migraine, there is a limitation of vision to one-half of the visual field, a hemianopsia; or the pain is associated with general obscuration of vision or certain visual hallucinations. In glaucoma there is an increased intra-ocular tension, and that instrument for the crucial test, the ophthalmoscope, shows the characteristic cupping of the optic disk.

With these preliminary remarks, let me proceed to the consideration of the various types of headache, *seriatim*.

(1) HEADACHE DEPENDENT UPON ORGANIC DISEASE OF THE BRAIN OR ITS MEMBRANES. The main characteristic of organic headache is the constancy of the pain. In this, it is different from all other forms. It may be, in fact usually is, subject to paroxysmal exacerbations, periodical or otherwise, but between the periods of maximum intensity, there remains more or less pain. An exception to the constancy of the pain in organic brain disease is found in the very early stages of such affections, when the pathological changes have not advanced to a degree sufficient to produce much irritation or pressure. The character of the pain varies in different cases; usually it is severe and agonizing and intensified by whatever causes that serve to increase the intracranial vascular supply, as stooping, exertion, and coughing. Cephalic sensations other than pain, as pressure, numbness, creeping, weight, heat, coldness, etc., are rarely symptomatic of organic intracranial disease. The pain may be limited to but a small area of the head, or it may be widely diffused. There is no essential relation between the location of the pain and the site of the lesion. There are, however, certain principles that aid us in a measure in establishing a relation between the seat of the pain and the lesion that gives rise to it. When the disease



is limited strictly to the meninges, the pain is almost invariably over the site of the lesion, and is associated with some local tenderness. The further removed from the meninges given focal lesions are, the less likely is the pain to be a guide as to its locality. Lesions beneath the tentorium generally produce occipital headache. Yet one will come across, on rare occasions, cases of cerebellar disease in which the entire pain was situated in the forehead. In fact, frontal pain may originate from lesion in any portion of the cranial contents.

The organic diseases producing headache have two pathological characteristics: They are capable of producing irritation or pressure or both. Irritation, however, is the main factor. In hydrocephalus, the disease of all others in which intracranial pressure is the greatest, pain may be entirely absent from the beginning. In meningitis and tumors adjacent to the membranes, when irritation must be great, the pain is correspondingly severe.

Frequent associations of organic headache, are nausea and vomiting (with clean tongue), fever, delirium, and optic neuritis.

In the classification of headaches at the beginning of this chapter, the organic diseases of which headache is a symptom were mentioned. In tumor, the pain is apt to be severe and paroxysmal. The intensity depends upon the proximity of the lesion to the meninges. Other things being equal, the larger the tumor, and the nearer it approaches the surface, the greater will be the pain. A lesion in the white matter (which by physiological experiment has been determined to be insensible) may be attended by pain. In meningitis, the pains are sharp and fixed. In cerebral abscess, the pain is not apt to be so severe as in the case of tumors of corresponding position and size; in abscesses from traumatism, the pain is not apt to be so great as in cases arising from ear disease. The latter are the larger, and are often accompanied by meningeal involvement, an all-sufficient explanation of this differential point.

Certain organic inflammations of the dura mater, as those arising from traumatism, tuberculosis, rheumatism, syphilis, and extension of disease through the cribriform plate of the ethmoid bone, may occasion headache. The pain under these circumstances is strictly localized, showing no tendency to shift from place to place. It is persistent, and seldom ceases for long at a time. It is accompanied by more or less mental depression and irritability, and disinclination for mental and physical labor.

(2) HEADACHES DEPENDENT UPON ALTERATIONS IN THE VASCULAR SUPPLY TO THE BRAIN. They may be either anæmic or congestive.

(a) *Congestive Headaches.* In this variety the pain may be either local or general, generally, however, the latter. In the former event, its point of greatest intensity is in the immediate vicinity of the points at

which the large veins enter the longitudinal sinus. The pain is accompanied by subjective sensations of heat, which are proven by means of the surface thermometer to have an objective origin. The patient's sufferings are aggravated by the recumbent posture, and by repeated acts of coughing. It is well to bear in mind that a cough may apparently of itself give rise to a very persistent headache. In fact, I have seen one case with the headache so much more prominent than the cough, that it was the patient's only source of complaint. The history of cough was only elicited after systematic search for the cause of the headache. Usually congestive headaches are associated with marked throbbing of the bloodvessels of the head and neck, and suffusion of the face. Da Costa has laid stress on the value of the ophthalmoscope in revealing the circulatory condition. I feel, however, that in this place, that instrument is not a reliable guide, for different observers, equally skilful, will present reports at wide variance with each other, when they come to describe the size of the retinal vessels. The action of the heart is greatly accelerated. The mental condition is one of irritability.

(b) *Anæmic headache* is especially apt to occur after hæmorrhages, as post-partum, in case of hæmorrhoids, and as an accompaniment of constitutional anæmic states. It is frequently met with in chlorosis, and Rake has described a case of pernicious anæmia (*British Medical Journal*, 1885, Vol. II, p. 913) in which it was the patient's only source of complaint.

The pain is usually symmetrical, and situated in the vertex. Sometimes it is located by the patient as immediately back of the orbit, and is described as if the "eyes were being drawn out of their sockets." It is greatly relieved by lying down. In severe cases, efforts to rise may produce syncope. The characteristic anæmic cardiac murmur is present.

### (3) HEADACHES ORIGINATING IN CERTAIN TOXÆMIA.

(a) *Uræmic Headaches*. Headache is not an infrequent concomitant of both acute and chronic uræmia. To the former it is a precursor, and lasts but a comparatively short time. It is then the result of any of the forms of kidney disease. As a symptom of chronic uræmia it exists mainly in connection with chronic contraction of the kidney. It may last for many months. The pain may be situated in almost any portion of the head, principally, however, in the frontal and occipital regions. It is, as a rule, quite intense; and it may be either paroxysmal or continuous. The diagnosis must be made by the urinary examination and the detection of symptoms significant of renal disease. One must not lose sight of the fact that the form of Bright's disease in which chronic headache is the most frequently met with, is also the one in which albuminuria is the least prominent. A quantitative analysis of the urine for urea should be instituted in every suspected case.

(b) *Lithæmic Headaches*. The headache of lithæmia apparently has no special diagnostic characteristics. Wood teaches that its usual

form is a dull and heavy pain ; while if we are to believe Haig, it usually assumes the migrainous type. The latter authority almost takes the ground that migraine and uric acid headache are synonymous terms. The paroxysms of pain recur at the intervals of a week or ten days, or from that to once a month. It is very rare that individual attacks last longer than from twelve to thirty-six hours. They are especially apt to be brought on by a highly nitrogenous diet, as butcher's meat and cheese, and are greatly relieved, if not entirely cured, by a strict adherence to a vegetarian existence.

The normal relation between the amount of uric acid excreted in the urine and the urea, is as 1 : 33. Any cause that serves to increase this ratio may excite the headache. But as Haig has shown, in order to have a plus excretion of uric acid, there must previously have been a storing up of this substance in the system, as shown by a diminished excretion, as 1 : 40. When the plus excretion comes, it is in exact proportion to the previous retention. Thus the urea-uric acid relation may be as high as 1 : 20, if the preceding retention warrants it. Haig places but little reliance on the actual quantity of uric acid, but only on its relation to the urea.

Accompanying the lithæmic headache is usually a high tension pulse. Preceding it, is usually a fit of mental depression. Some observers have reported cases in which the opposite mental condition prevailed, the patient being unusually happy.

On the days preceding the headache, the urea-uric acid relation is diminished ; on the day of the headache, it is greatly increased.

The uric acid headache may begin in childhood, owing to hereditary predisposition, or to improper diet.

The gouty headache is but a variety of the lithæmic, in fact an advanced stage of the latter. In this case, we find ample evidence of gout elsewhere. The headaches are relieved whenever other portions of the body are the seat of gouty invasion. It has been claimed that the headaches of gout are in reality of the organic variety, being the result of changes in the brain and its membranes from the patient's dyscrasia.

(c) *Rheumatic headaches* are usually of a dull, heavy, aching character. They are especially apt to be associated with soreness of the scalp, and are aggravated during spells of damp or cold weather. In occasional instances, they may be the result of rheumatic inflammation of the nerves of the scalp, in which case the pains will partake of the neuralgic character.

Occasionally one meets with a type of rheumatic headache described by Nordstrom. The pains partake of the character of those of migraine. Investigation, however, shows that they depend upon thickening of the insertions of certain muscles, notably the sterno-mastoid and occipito-frontalis. These indurations do not usually give rise to local pain ;



hence they are apt to escape observation. They are commonly the result of taking cold. Nordstrom finds an efficient remedy in massage of the indurated spots.

Along with the headaches of septicæmia, may be mentioned that ushering in typhoid fever, and other low forms of acute disease. As already stated, the fact must simply be borne in mind that such headaches may occur as prodromal symptoms without other evidence of subjective disease.

(d) *Diabetic headaches.* Headache does not appear to be an especially annoying symptom during the ordinary course of diabetes. In the later stages of the affection, when acetonæmia supervenes, it is of great severity, and of especial importance in view of the probable onset of diabetic coma. Under these circumstances it is apt to be associated with epigastric pain, gastric disturbance, vomiting, dyspnoea, and delirium.

(e) *Malarial headaches.* Head pains due to malaria *per se* are characterized by their periodicity. They are usually situated near one or the other supraorbital foramen, and come on at a fixed hour every day or every other day. They may or may not be accompanied by other evidences of malaria. Sometimes the pain takes the place of one of the typical stages of intermittent fever, chill, fever and sweat.

We sometimes meet with secondary malarial headaches, that is those dependent upon the concomitant gastric disturbances and anæmia of intermittent fever. In such cases, the pain does not present the periodicity referred to.

(f) *Syphilitic headaches.* A severe form of headache is often met with during the secondary stage of syphilis. The pains are of a boring and splitting character. It generally involves but a portion of the head, and is decidedly worse at night. There is considerable tenderness of the scalp. The headaches of tertiary syphilis are due rather to organic lesions than to any special toxæmia. Syphilitic headache may occur as early as six weeks after infection.

(g) *Headache from gastric disorder.* The majority of pains in the head in conjunction with dyspepsia are of a purely reflex character. There still remains a number of cases in which the difficulty arises from decomposition of food in the intestinal canal, and the absorption of the products. The pain in such cases may be very severe, as in a case reported by me in the *Hahnemannian Monthly* for 1886. In this case, so great were the patient's sufferings that organic brain disease was diagnosed. The severe vomiting and the horribly putrid breath suggested a retention of food and decomposition of the same in the digestive tract. Washing out of the stomach with gavage was practised on alternate days. In ten days' time, all pain and vomiting had ceased, and the tongue had become clean. After several months' steady treatment the patient was practically cured aside from an atrophy of the optic nerve which progressed to complete blindness.

Headache is a very frequent symptom from constipation. This point is one that is altogether too much neglected by members of our school. It is not at all singular that the retention of such foul substances as faecal masses within the human organism any longer than normal should cause trouble. The only wonder is that carelessness in attention to the bowels does not occasion more difficulty than it does.

(h) *Toxic headaches.* Of poisons introduced into the system from without, alcohol is the one that most frequently produces headache. It does this not only by its effects on the blood, but also reflexly by first exciting gastric disorder.

Other poisons, as lead, strychnine, quinine, and the narcotics, opium, chloral, etc., may also produce chronic headaches. No special phenomena are attached to the pains from these causes. The diagnosis must be based on a knowledge of the habits of the patient, together with a study of the associated symptoms.

(4) HEADACHES REFLEX FROM DERANGEMENT OF ADJACENT OR DISTANT ORGANS. (a) *Ocular Headache.* Ophthalmic difficulty is a very frequent cause of chronic headache. The affections active in this respect are errors in refraction and insufficiencies of the ocular muscles. The former are more frequent causes, simply because they are the more common. Certain forms of muscular insufficiency are almost certain to produce headache. Astigmatism is the most frequent and active refractive error in the production of headache; compound hypermetropic astigmatism is the most important in this respect; simple myopia is the least likely to give rise to such difficulty. Of the muscular insufficiencies, hyperphoria is almost certain to produce headache, not more than 10 per cent. of those subject to it escaping. The degree of refractive error or muscular insufficiency does not seem to bear a definite relation to the severity of the pains. I have seen long-lasting headaches radically cured by the correction of a simple hypermetropic astigmatism of 0.25D; and I have seen cases with a comparatively high degree of refractive error causing only visual disturbance. If there is any rule, it is to find the headaches in persons whose acuity of vision is normal, and in whom there is but a low degree of ametropia. The same remarks apply to muscular insufficiencies. If there is a high grade of weakness the patient soon learns to disregard the weakened eye, and makes no effort to overcome the difficulty.

The pain in cases of errors in refraction is usually found in the frontal region or in the eyeballs. That from muscular insufficiency is located generally in the cervical region or in the occiput. The pain may be either dull or acute; constant or paroxysmal; or, again, it may partake of all the characters of a typical migraine. While in the majority of cases the pain is much aggravated by the use of the eyes, still cases are not infrequent in which the symptoms do not offer the slightest sug-

gestion that the eyes are the seat of the difficulty. Then the only test is a thorough ophthalmic examination.

Difficulties in diagnosis are presented because of the frequency with which these headaches occur in neurotic subjects. A very large percentage of mankind is ametropic, and yet but comparatively few of these suffer in consequence. It seems that a certain amount of neurotic constitution must be present. Some ametropic patients go on for years without any trouble whatever; and then after a severe illness, or great mental or nervous strain, symptoms appear. Such cases require, after correction of the refractive error, most careful attention to the condition of the general system.

In extreme cases, eye-strain may occasion not only headache, but even nausea, vomiting, vertigo and insomnia.

(b) *Headaches dependent upon Disease of the Nose and Naso-Pharynx.* The well-recognized industry of the oculist has made the existence of headaches from eye-strain a well-attested fact by all physicians. The rhinologist has not impressed the importance of his domain in this respect upon the profession. This, we think, is unfortunate, for nasal headaches are not uncommon; and when they do occur, are apt to escape recognition. I well remember my first case of this character. There was well-marked conjunctival irritation; so I corrected the patient's refraction without benefiting him in the least. Because of a nasal obstruction, I examined his nose and found a polypus, the removal of which made a radical cure of the headache. Not only may headache arise from nasal disease, but also from certain affections of the nasopharynx, as enlargement of the pharyngeal tonsils, follicular tonsillitis, hypertrophied tonsils and obstruction of the Eustachian tubes. Harrison Allen has described three forms of nasal head-pains, as follows: The reflex, the neurotic and the inflammatory. "The reflex headache is almost entirely restricted to the forehead, the temple and the vertex. By drawing the index finger across the face from the middle of the nose to the temple, and thence, in some cases, to the parietal eminence, the patient often indicates the seat of the pain. In severe attacks, pain sometimes radiates to the vertex, and even to the nape of the neck, and then often nauseates and simulates migraine. Sometimes the point of pain is narrowed to a minute focus or spot. A very characteristic symptom is the marked increase of the headache upon the slightest exacerbation of the catarrh. A diagnostic symptom is tenderness of the inner wall of the orbit when pressed upon by the finger; or a probe passed into the nose causes an immediate access of pain when it reaches the right middle turbinated bone." The disappearance of the catarrhal reflex headache, when the nasal catarrh is cured, is the strongest proof of its nature. The neurotic nasal headache of Prof. Harrison Allen comprises cases in which highly neurotic individuals complain of pain



in the throat, the ears and back of the head, or even in the pharynx, or of various distresses about the head, as a result of moderate degree of local nasal or pharyngeal disease. Dr. Allen further says that he has never seen catarrhal headache of inflammatory origin, except in acute congestions or inflammations of the frontal sinuses; the pain is of a high grade; is, as a rule, confined to one side, and subsides after the local application of leeches.

(c) *Headache from Ear Disease.* The fact that chronic aural affections other than suppurative may occasion headache is not generally recognized; yet such cases do sometimes occur. I have seen one such in which the pain was of a dull character, more or less constant, and situated just above the affected ear. There was no localized soreness, and other than slight hardness of hearing there were no aural symptoms. According to Dana, middle ear disease may cause temporal and occipital pain.

(d) *Headache Reflex from Gastro-intestinal Disorder.* I have already stated that toxic headaches may occur in connection with digestive disturbances. The same is true as regards the production of reflex disturbances.

(e) *Headache Reflex from Disease of the Sexual Organs.* Disease of the sexual organs in both sexes may occasion headache. In the case of the male, the difficulty is apt to be neurasthenic; in the female, however, it is a true reflex. The pains in the latter case are almost invariably situated in the vertex or in the occiput.

(5) NEUROPATHIC HEADACHES. (a) *Epileptic headaches* are often spoken of, and yet I doubt if such, aside from being an occasional sequel of convulsive seizures and the still more rare occurrence of epileptic migraine, are ever seen. Dana describes what he calls an epileptic headache by saying that the pains are usually sharp and severe, often vertical or occipital, but not diffused. He says nothing about the circumstances under which the attacks appear. Gowers refers to a kind of headache which all having any experience with epilepsy have probably observed, coming on immediately after the patient awakes from the post-epileptic sleep. The pain is general and lasts for several hours or more. Hare describes what has been called epileptiform migraine. "In these cases there is flexion of the fingers of both hands and numbness of the feet, with violent pain in the head of a hemicranial character. The tongue feels too large for the mouth, and speech is difficult. When writing is attempted (for consciousness is preserved), although words are ready to flow, only a meaningless scrawl results. The eyesight grows dim and the pain in the temples increases in violence; these symptoms being followed by vomiting and deep sleep, from which the patient awakens well. As the attacks go on, consciousness becomes slightly dimmed; but is never lost until the disease is very far advanced."

(b) *Hysterical Headaches* characteristically assume the form known

as clavus, in which the pain is limited strictly to one spot in the head, a spot that may almost be covered by the tip of the finger. It is apt to be relieved, if not entirely driven away, by excitement or matters that appeal strongly to the patient's interest.

(c) *Neurasthenic Headaches* are met with in persons who are "run down." The pain may be constant or deep-seated; or it may consist of certain indefinable disagreeable sensations. It is greatly increased by mental effort. Like the hysterical headache, it may disappear almost completely under excitement.

Many of the periodical headaches with which children are affected are of this type, and are due almost exclusively to the persistency with which those of a neurotic type are forced in their studies. I might say that almost all the headaches of children are due either to refractive errors or to overtaxation of the brain.

(d) *Migraine* is such an important affection that I shall devote the succeeding chapter to it.

(e) *Essential Headaches*. Occasionally one meets with patients who are subject to headache of such a character and under such circumstances as to make it utterly impossible for the physician to determine their causes. Those I have seen have mostly been such as to make me believe that they were maintained by attention. Treatment availed nothing; even the usual palliatives failed to palliate.

## MIGRAINE.

**Synonyms.**—Hemicrania; megrim; migrän (German).

**Definition.**—Migraine is a disease characterized by paroxysms of headache, generally associated with vaso-motor and sensory symptoms; though exceptionally, the attacks consist of the sensory phenomena mainly or entirely.

**Etiology.**—Migraine may be said to be a disease of civilization. No neurosis numbers among its victims so many eminent scholars. It unquestionably occurs with greater frequency in persons of neurotic constitution. Women, in particular, are predisposed to it, about two-thirds of all cases occurring in the female sex. Hereditary influences are very powerful factors in its production. While in the majority of cases the transmission is a direct one of migraine from parent to child, there are many instances in which one neurotic manifestation in the parent, *e. g.*, migraine, is transmitted to the child as epilepsy or hysteria. The disease generally begins in early life, the majority of the patients experiencing their first attack between their fifth and tenth years. Cases have been known to occur at even an earlier age. The more markedly operative the hereditary influence in a given case, the earlier in life the disease is likely to begin. The liability to the onset of this disease is greatly lessened after the age of thirty years. Some few cases set in, however, even up to quite advanced years, *e. g.*, the age of sixty. Gouty and rheumatic constitutions are not infrequent subjects of this disorder. Haig makes the claim that the uric acid diathesis is the all-powerful etiological factor in migraine. He is undoubtedly right in many instances; but his conclusion that the majority of cases are of the lithæmic class is altogether too sweeping, and is not warranted by the clinical data at our disposal.

Reflex irritation has been alleged to be a frequent cause of this disorder, the eyes, nose, and teeth, coming in for blame. Undoubtedly errors in refraction are sometimes productive of paroxysms of this painful affection; but in the majority of cases, they cause less severe headaches. Anomalies or insufficiencies of ocular muscles must also be borne in mind as a cause of reflex head pain. Nasal and pharyngeal disease have occasionally been active causes, and Brunton has met with cases dependent upon diseased teeth.

As a rule, the paroxysms themselves occur spontaneously without any exciting cause. By long experience, patients learn to recognize the influences which act deleteriously, and avoid them. These exciting



causes vary in different individuals. Some persons are comparatively free from trouble until they experience severe mental and bodily fatigue; in others the headache appears at the call of powerful depressing emotions; some persons suffer after a long railway journey; many women have attacks bearing some definite relation to the menstrual periods. But in most cases, as already stated, no exciting causes are manifest.

**Pathology.**—Most carefully conducted investigations have thus far failed to reveal any structural alterations as the basis of migraine. The pathology of the disease must, therefore, be based entirely upon speculative considerations. Two theories have been advanced in explanation. The one having most to commend it, is that advocated by Gowers, to the effect that the attacks are dependent upon periodical discharges of certain sensory cortical cells in the brain, in other words a “nerve storm.” This theory brings migraine into close relationship from a pathological standpoint with epilepsy. The other theory assigns a vaso-motor origin to the disease. It provides that at the beginning of the attack there is vaso-motor spasm, and that this is followed shortly by vaso-motor paralysis. Many authorities accepting the vaso-motor theory, divide cases of migraine into two classes, one dependent upon vaso-motor spasm, and the other upon vaso-motor paralysis—angio-spastic and angio-paralytic, respectively. This division has been made the basis for prescriptions of such drugs as glonoin, ergot, etc.

The old notion that the attacks are of gastric or hepatic origin has long since been exploded. The only ground on which such a notion ever existed was the severe vomiting, often of bile, with which attacks passed off.

**Symptoms.**—As already intimated migraine consists of paroxysms of headache associated with quite a variety of vaso-motor and sensory phenomena. The attacks themselves are usually ushered in by certain prodromal symptoms. Sometimes there may be heaviness in the head, drowsiness, or a slight pain, appearing the day before the paroxysm. Immediately preceding the headache, in very many instances, are visual disturbances. These vary greatly in character in different cases. Frequently there is noticed a dimness of sight on one side, and this gradually increases to complete hemianopsia. In still other cases there may be blind spots (scotomata) or luminous spectra in the field of vision. The visual hallucinations oftentimes assume most grotesque appearances, thus lightning-bright lines shaped like “walls of Troy” are described; sometimes the patient tells of many saw-teeth of fire appearing before him, and these take on a rapid to and fro motion. The “visions” may assume even more definite shapes, and the patient sees animals, objects or persons. The hemiopic character impresses itself upon nearly all these visual symptoms.

Vertigo is often experienced. It may appear early; usually, how-

ever, it is deferred until after the onset of the sensory disturbances. Tinnitus aurium and deafness are less frequent than the ocular disturbances. Affections of smell and taste are very unusual, though perfectly possible. In some cases the pupils exhibit most remarkably rapid changes, that of alternate contraction and dilatation, known as hippus.

Sensory symptoms consisting of numbness and tingling, and referred to various portions of the body, are also experienced. Usually they are felt in the extremities, less frequently in the face and tongue. In very rare cases, transient motor or sensory aphasia is an interesting phenomenon. With this accompaniment, the sensory symptoms in the extremities are referred to the right side. Occasionally the sensory phenomena are succeeded by motor weakness of the same parts, thus suggesting a resemblance to certain cases of epilepsy.

Mental excitement or mental depression may precede or accompany the seizures in many instances. The patient may exhibit confusion of ideas, or a stuporous condition. The frequent occurrence of such attacks may lead to permanent mental failure.

The headaches themselves follow very shortly on the above described sensory symptoms. The pain begins in one small spot, or on one side, and rapidly spreads until finally it may involve the entire head. More than that, it may extend into the neck and arms in very severe cases. It is of agonizing character, being described by the patient as sharp and boring or throbbing. It may begin mildly and gradually increase in intensity, or it may reach its acme shortly after its onset. It is very much intensified by the slightest peripheral impression, light, sound, and jars, all alike producing aggravation. The attacks are very prostrating, the weakness sometimes amounting to almost absolute paralysis. Individual cases present peculiarities in the course pursued by the pain and its attendants, these peculiarities being the same in all paroxysms occurring in the same individual.

Vaso-motor symptoms in the shape of pallor of the face, retraction of the eyes and contraction of the pupils, accompany the headache. These symptoms may be succeeded by flushing of the face, and the pupil, at the same time, regains its normal size. There may be cedema of the scalp and unilateral sweating.

The patient has little or no appetite throughout the seizure, nor is it desirable that she should have any, for most of the food taken is vomited, and that which is retained is not digested. Usually the headache goes off with nausea and vomiting. The latter may be frequently repeated until finally there is vomiting of bile. Other attacks terminate by profuse urination or perspiration. The most frequent termination is by a long, refreshing sleep, from which the patient awakens free from pain.

These phenomena constitute the typical attack of migraine. We

may meet with cases in which the sensory symptoms constitute the entire attack. More rarely, paroxysms of acute mania are substituted for the pain. Thickening of the coats of the temporal artery and marked arcus senilis have been observed in a few instances on the side corresponding to the pain.

Galezowski has described a form of hemicrania which he has called ophthalmic migraine, and in which paralysis of certain ocular muscles comes on with the headache, and persists for some days. This may be confounded with syphilitic ophthalmoplegia; but the latter comes on gradually. If headache accompanies the attack, it both precedes and accompanies the palsy to its finale.

It is interesting to note that migraine may alternate with other neuroses, such as epilepsy, neuralgia, etc., in the same individual.

The temperature is rarely altered. In children, however, the paroxysms may be accompanied by quite marked rises in temperature.

The attacks vary in duration from five or six hours to three days. They recur generally at intervals of from two to four weeks. Women are especially liable to experience a recurrence at the menstrual period.

**Diagnosis.**—There is danger only in confounding migraine with other varieties of headache. All error may be avoided by attention to the periodicity of the affection, and the association of the pain with the peculiar sensory phenomena. It is important to remember that the same patient may have migraine at one time, and a headache dependent upon some entirely different influences at another.

When the sensory or vasor-motor phenomena occur without headache, there is a liability of mistaking them for seizures of petit mal. The duration of the attack is here our main reliance; that of petit mal is rarely for more than a few seconds, while that of migraine is never less than from twenty minutes to half an hour.

**Prognosis.**—The disease is not at all dangerous to life. The prospects of an absolutely complete cure are, however, rather small. Some authorities make the claim that migraine is unquestionably incurable. In the majority of cases there is a spontaneous cessation of the disease at middle life or shortly after. Proper methods of living will do considerable towards lessening the frequency and severity of the seizures.

**Treatment.**—In the way of general management, the patient should be directed to avoid all excitement, and observe regularity in all his habits. Excesses of all kinds and the use of tobacco, alcohol, etc., must be discontinued. The greatest care should be taken with the bowels, which ought to be habituated to regular daily action. Children should not be permitted to overtax the brain with study, and above all things, competition for prizes for proficiency in scholarship must not be permitted. All sources of reflex irritation, as refractive errors, nasal disease, etc., must receive attention.



During the paroxysm, the patient should be put to bed. The diet should be of the lightest character possible. It is a matter of small importance if the patient fasts, unless the paroxysms are very frequent and long lasting. For relief of the pain, the coal-tar derivatives may be appealed to. Antipyrin seems to be the most efficient, but it also requires more care in its administration. I have derived most satisfaction from a mixture of acetanilid and caffeine in the proportion of three and a half grains of the former to one-half grain of the latter, four to eight grains of the mixture being given as the initial dose. The smaller dose may be repeated in one hour, but not again until the patient's susceptibility to the drug is positively known. Phenacetin in ten grain doses is a very efficient palliative in many cases. The above-mentioned drugs are generally regarded with some suspicion, owing to the damage they have done when incautiously or indiscriminately used. Prescribed with care, they accomplish much in the way of palliation. They are far superior to morphia, which has the positive disadvantage of bringing on the morphia habit. Some patients find in an effervescent solution of bromosoda with caffeine a most efficient remedy. In some few cases the repeated administration of acetanilid and its congeners, by palliating the pain decidedly lessens the frequency of the paroxysms.

Massage was used by Nordstrom in cases in which examination showed subcutaneous thickenings, tenderness, or irregularities in the scalp muscles. These cases have no hemianopsia, scotomata, or other symptoms characteristic of the true migraine.

Recent cases call for *belladonna*, *gelsemium*, *calcareea*, *ignatia*, *spigelia*, *nux vomica*, *sepia*, *silicea*, and *stannum*.

*Belladonna* is suited to young slender subjects, of nervo-sanguine temperament. There is the characteristic throbbing pain, flushed face, aggravation from dependent position of the head, and other well-known symptoms.

*Sepia* and *calcareea* are valuable remedies, indicated, however, on constitutional conditions rather than on any specific characters to the pains. The *calcareea* patient is of a lymphatic constitution. The *sepia* patient is usually a woman, of sallow complexion, subject to uterine disturbance and leucorrhœa. The pains are of a throbbing character, and shoot upward or from within outward. There may be a jerking of the head backward and forward.

*Ignatia* is the remedy for hysterical patients, especially when the pain assumes the characteristics commonly known as *clavus*. The attacks are brought on by emotional influences. Slight clonic spasms of the hands may accompany the paroxysms, which generally pass off, accompanied by a free flow of urine. The face is pale during the attacks.

*Stannum* is a favorite remedy with Hughes. Its pains commence gradually, and disappear as gradually as they came.

*Sanguinaria* and *iris versicolor* are the favorite remedies of most practitioners for the attacks themselves. They are also valuable remedies to be administered in the interim as curative agents. Under *sanguinaria* the attacks recur periodically, the pain commencing in the occiput and settling over the right eye, with nausea and vomiting. Sometimes the pains begin in the forehead and vertex, on the right side. Usually the attack begins in the morning, and lasts until evening. It obtains marked, if not complete, relief by sleep. As under *ignatia*, it often passes off with free flow of urine.

*Iris* was the favorite remedy of Dr. James Kitchen. The pains are situated mostly in the forehead or right side of the head, and are aggravated on beginning to move, and relieved by continued motion. They are associated with lowness of spirits, nausea and vomiting. Vision is nearly always disturbed. Both *sanguinaria* and *iris* are often prescribed empirically.

*Sulphur* is recommended by Hughes as the remedy for cases of migraine occurring in patients of gouty diathesis.

*Cannabis indica* is highly recommended by Hale when the attacks recur periodically every week or two. The pains are agonizing, and the patient delirious or unconscious at times. The attacks prostrate the patient greatly. The face is pale; the head cool; light and noise aggravate the pain. No vomiting occurs. This remedy is the favorite old-school remedy at the present time. It is administered in quarter of a grain doses of the extract, three times daily; this dose to be gradually increased until the limit of the patient's susceptibility is reached. It is claimed by Seguin and others that the tendency to recurrence is thus greatly lessened.

*Glonoïn* is certainly a valuable remedy in some cases, especially when the disease is associated with marked intra-arterial tension.

Electricity is of aid in some few cases. Static electricity, applied by the soufflé to the head, has cured or relieved quite a number of patients. Galvanism to the head has accomplished but little beyond slight temporary relief.

## NEURALGIA.

To accurately define the term neuralgia is not an easy matter. In general, we use it to designate those cases of nerve pain occurring independently of pathological changes in the nerve trunks themselves, as neuritis and neuromata. We exclude from the category of neuralgia pain in the distribution of a nerve arising from the mechanical effect of disease in adjacent tissues.

**Etiology.**—The great predisposing cause of neuralgia is the neuro-pathic constitution. Nervous, excitable patients, especially those made so by hereditary transmission, are liable to neuralgic pains under the influence of the exciting causes to be mentioned, and sometimes without any exciting cause whatever. The typical neuralgic patient is generally found depressed in other directions also. Thus anæmia, general exhaustion, as from overwork and excessive lactation, are by no means infrequent causes. General blood states, as gout, rheumatism, malaria, diabetes, lithæmia, often predispose to it.

The disease is essentially one of adult life, children being almost entirely exempt. It is very rare in old age, that is, after the sixtieth year of life, except as evidence of oncoming degeneration.

Almost any deleterious influence may excite neuralgic pains in those predisposed. Exposure to cold is not uncommonly one of these, as are excitement, emotions of any variety, and mental and physical overwork.

It has often followed immediately on certain of the acute infectious diseases, notably influenza.

*Reflex causes* frequently are operative in producing neuralgic pains. Prominent among these stands dental disease as a cause of facial neuralgia. It is not necessary that the pain shall be in the area supplied by the nerve branch going to the affected tooth. Indeed, widespread pain may be occasioned without the slightest discomfort at the source of the trouble. Neuralgia even extending into the arm and neck has been known to follow dental caries.

*Traumatic agencies* undoubtedly cause nerve pains in many instances; generally, however, by producing alterations in the tissues. Possibly the traumatism sometimes acts as a systemic depressing agent.

Neuralgias have been known to occur as the direct result of certain oft-repeated movement or act in following one's occupation. This act may be such as to produce a local tire, or a local traumatism.



*Hysterical* neuralgias occur in neurotic individuals, and are generally associated with the so-called sensitive spine. These patients are often anæmic. Pains in the joints and ovaries are very common.

A variety of neuralgia has been described as epileptiform, from a theory as to its origin. It affects the trifacial nerve solely.

**Symptomatology.**—Individual cases of neuralgia present the greatest differences in their clinical histories. The general characteristics are attacks of pain of very variable duration, coming on suddenly or gradually, at variable intervals, which may or may not exhibit a regular periodicity. The pain is almost always limited to one side of the body. It may, however, be bilateral, in which case it is almost always symmetrical. It may, in individual cases, always attack the same area, or it may exhibit a disposition to attack widely separated parts at different times. The intensity of the pain is very variable. In one attack it is unbearable; during another it causes but little more than the slightest discomfort. Even during a paroxysm the pain is often characterized by its fluctuations in intensity. The character of the pain presents great variations. It may be dull, sharp, darting, throbbing, stabbing, etc.

Paroxysms may appear spontaneously or in response to certain extraneous influences, as noises, light, warmth, movement, or emotions. During their continuance the patient is often hyperæsthetic to all external impressions, which may even cause exquisite pain. Sometimes the pain is preceded by sensations of numbness and tingling in the parts about to be affected.

In very many old standing cases of neuralgia sensitive spots, so-called, are found. These were first described by Valleix, and have been named after him. These sensitive spots correspond to points at which the nerve trunks emerge from bony canals, over a hard structure, or through a fascia, to become superficial. Neuralgia limited to each particular nerve has its own fixed sensitive points. Very often one finds sensitiveness of the vertebra corresponding to the nerve affected.

Vaso-motor and trophic phenomena occur in many cases. These include alterations in the circulation, as pallor and flushing, changes in the hair and local malnutrition.

**Diagnosis.**—The diagnosis of neuralgia is to be made by the unilateral character of the pain, its limitation to the area supplied by special nerve trunks, the intermitting character of the pain, and the absence of symptoms pointing to other conditions. The greatest liability to error lies in confounding it with *neuritis*. In the latter affection the pain is more constant, and the functions of the nerve are quite considerably interfered with. Thus anæsthesia and paralysis are prominent symptoms as a rule. The nerve trunk is more tender and the pain is far more constant.

Very often neuralgia has been confounded with the *lightning pains*

of ataxia. The latter are always found associated with other symptoms pointing indisputably to organic changes in the spinal cord.

*Visceral neuralgias* very closely simulate organic disease of the different organs. A differentiation is only possible by careful and repeated physical examinations, and attention to the clinical course of the case over a long period of time.

**Prognosis.**—Owing to the many points to be taken into consideration in the clinical study of neuralgia, it is impossible to lay down any hard and fast rules governing the prognosis. Each case must be studied on its merits. If the etiological relations of the case are clear and the cause is removable, the prognosis is correspondingly favorable. Advanced years lessen the prospects of recovery. A marked hereditary neuropathic tendency makes the prognosis correspondingly unfavorable.

**Treatment.**—The successful treatment of cases of neuralgia requires a careful study of the neuralgia itself, and also of the associated conditions. The latter in many instances, if not in nearly all, furnishes the most important indications for treatment, both hygienically and medically. In every case the cause of the disease must be sought and removed. The general standard of health must be raised to the highest possible. Good food, fresh air, and mental and physical rest are necessary in many cases. Sometimes it is important that the patient be sent away from home for a more or less protracted period. The diet of neuralgic patients must not be prescribed on an empirical basis. Anæmic and neurotic cases require liberal feeding, the main portion of the food being of a fatty and animal nature. If the patient cannot partake of fat food, then it should be supplied in the shape of cod-liver oil. Lithæmic patients require abstinence from meat, and free indulgence in pure water. Cases dependent upon diabetes or renal disease require the management indicated by these troubles.

Rest of body and mind is of the highest importance, especially in the overworked and the troubled. Mental rest is to be secured by diversion of the mind to other than the subjects over which it is usually occupied, and is always a difficult matter to secure. Physical rest should be graded according to the case. Some cases are best treated by absolute rest in bed for a protracted period; others require simply a daily rest of an hour or two.

For the relief of the pains during the paroxysms, the best palliative in many cases is dry heat. This may be applied by means of the Japanese fire-box or the hot-water rubber bags, or hot hop-bags. These often fail to give the desired comfort, and other palliatives become advisable if not absolutely necessary, for long-continued pain is exhausting. Morphia should never be used excepting as a last resort, because of the danger of forming the morphia habit. Above all things, the patient should never be intrusted with this drug, especially if it is to be

administered hypodermically. If an analgesic is to be employed, it should be one of the much-abused coal-tar preparations, antipyrin, phenacetin, or acetanilid. These are all efficacious in pains about the head. Antipyrin is the most efficient in other pains, although acetanilid is but little inferior to it, and probably safer. While thus speaking in praise of these drugs, I do not advocate their indiscriminate exhibition. If the physician understands their action and doses thoroughly, he need not fear evil results; but let him give them recklessly, and he exposes his patient to dangerous risks.

Electricity is very valuable in many cases. Galvanism is the preferable current. The positive electrode should be applied over the sensitive points, and the negative over the spinal column at the supposed origin of the affected nerves. The current should be free from interruptions and inequalities. Its strength will vary according to the sensibility of the affected part, all the way from five to thirty-five milliamperes.

The surgical treatment of neuralgia resolves itself into nerve stretching and nerve resection. Relapses are very common after both procedures, the latter generally affording the more permanent results. Still stretching is often of service, and being a more conservative procedure, should be the operation of first resort.

The medicinal treatment of neuralgia is not easy to expound because of the many collateral conditions to be considered as indications for drugs. The following suggestions are offered:

*Actea racemosa* is valuable in many cases of rheumatic origin, and also in cases occurring reflexly from utero-ovarian disease. The pains are of a sharp lancinating character. They are usually supraorbital or intercostal. Still the pain may be referred to any of the peripheral nerves.

*Belladonna* is useful in quite a variety of neuralgic pains, characterized especially by their sudden onset, and the high degree of attendant congestion. The characteristic sensation is that of throbbing. The patient is mentally irritable. Hughes looks upon this remedy as limited in usefulness to recent cases.

*Arsenicum* enjoys a good reputation in both schools of medicine as an anti-neuralgic. In the first place, it is invaluable in neuralgias of malarial origin, especially when characterized by periodicity in recurrence. The pain usually affects one side of the face. The pains are worse towards night, reaching their climax at or after midnight. The patient is unusually restless. *Cedron* is another remedy for malarial neuralgia. The pain is supraorbital and is remarkable for the regularity of its occurrence.

*Mezereum* is probably the best remedy for neuralgia reflex from decayed teeth. In these cases, the importance of referring the patient to the dentist for operative treatment must not be forgotten.



Other remedies to be considered in individual cases are *rhus*, *bryonia*, *colchicum*, *chamomilla*, *spigelia*, *thuja*, *colocynth*, *phosphorus*, *sepia*, *cinchona*, *aconite*, and *sulphur*. Indications for most of these remedies will be found in the chapter dealing with general considerations pertaining to nervous diseases. To this the reader is referred.

## OCCUPATION NEUROSES.

**Synonyms.**—Professional dyskinesiae; anapeiratic paralyses (Hammond); professional neuroses.

**Definition.**—Under the head of professional neuroses is included a large class of nervous affections characterized for the most part by spasm or inco-ordination of certain muscles used habitually in the pursuit of the patient's occupations; said spasm or inco-ordination occurring, as a rule, when the act which gave rise to the trouble is performed, and not during other movements of the muscles, no matter how complicated they may be.

Of the occupation neuroses those best known are writers' cramp, telegraphers' cramp, musicians' cramp (violinists and pianists especially), and sawyers' cramp. Writers' cramp may be taken as typical of them all, it differing from the others only in the distribution of the affected muscles.

### WRITERS' CRAMP.

**Synonyms.**—Scriveners' palsy; graphospasmus; chorea scriptorum.

**History.**—This affection was first described by Sir Charles Bell in 1830. Its existence during the last century is decidedly problematical, for we fail to find mention of it in a work by Ramazini on the diseases of tradesmen. It is generally believed that the introduction of steel pens had much to do with its appearance.

**Etiology.**—The principal predisposing cause of writers' cramp is found in the possession of a neuropathic constitution. Very many of the cases will give a history of other neuroses, as epilepsy, chorea, neuralgia, and insanity, in near relatives. Some of them, indeed, will be found to have suffered from other nervous disturbances. The neuropathic constitution is by no means necessary, for the disease will occur in persons who are in possession of most robust health.

In keeping with the neurotic origin of writers' cramp and other occupation neuroses, are other causes of these disorders. Thus we often find long-continued anxiety operating to produce them. It will be noted that the patient performs his accustomed duties for years; a period of anxiety occurs, and symptoms of the disease set in, but disappear promptly on the restoration of the patient's ordinary tranquil condition. Depressing emotions, and causes which act to lower the general health standard, may be considered as frequently operative. Excessive indulgence in alcohol, tobacco and venery are prolific predisposing causes.

Some cases seem to find their origin in a local injury, and exposure to cold.

Be the predisposing causes what they may, the exciting cause is the abuse of writing. I have used the word "abuse" rather than "excess," because writers who write properly are unlikely to be affected. Those who make it a practice to write with the fingers and hands cramped, using the muscles of the hands and fingers only, are almost the only ones liable to writers' cramp. Those who write a free hand, bringing into action the large muscles of the arm and shoulder, almost entirely escape. Copyists are far more liable to it than are those who compose while they write (*e. g.*, authors, clergymen, etc.), probably because the latter rest the hand more while working. Writing under pressure in order to accomplish a set task within a given time is undoubtedly a prolific cause of the disease under consideration.

The character of the pen and penholder, the position of the desk and the sheet of paper, and the position of the body during the writing, are all important etiological factors.

**Pathology and Morbid Anatomy.**—The occurrence of peripheral neuritis in some cases of writers' cramp is undoubted; but it is equally certain that this pathological change is simply an accidental or associated lesion. In the majority of cases absolutely no morbid change is detectable in any portion of the nervous system. All assertions as to the pathology of the affection must therefore be based upon speculation. The most reasonable theory offered is that the disease results from the derangement of the nerve centres presiding over the muscular movements involved in the act of writing. In order that such highly complicated movements as those involved in writing shall be performed accurately and with fluency, there must be an education of nerve centres, some of which may be widely separated from each other. This is accomplished by the lessening of the lines of resistances between them, "so that the movement which was at first produced by a considerable mental effort, is at last executed almost unconsciously. If, therefore, through prolonged excitation, this lessened resistance be carried too far, there are an increase and irregular discharge of nerve energy, which give rise to spasm and disordered movements."

**Symptomatology.**—The onset of writers' cramp may be either gradual or sudden, the former, however, being the case in the vast majority of instances. Usually the patient notices that he does not grasp his pen with his accustomed readiness, and this leads him to hold it all the tighter. Gradually this difficulty increases, and he, to obviate it, adopts quite a variety of devices in the way of unusual positions while writing. Finally there occurs spasm of the muscles concerned in pen prehension, as soon as that instrument is taken in hand; consequently it slips from between the fingers. The spasm is always of a tonic character, and may



be associated with some tremor. This latter is always of the intention variety, it occurring only during the act of writing. It is finer in range than the so-called tremor of disseminated sclerosis. During the act of writing the pen is jerked about, imparting to the chirography the most irregular characteristics. Strokes are coarse and imperfect, frequently resembling zig-zag lines rather than written characters. The most careful examination fails to discover any weakness of the affected muscles unless neuritis be present.

Sensory disturbances are by no means unusual. Of these, pain is the most frequently encountered. This, at first, consists only of a sense of tiredness coming on shortly after beginning to write; but gradually increasing in severity as the disease advances, until finally it assumes a severe neuralgic character. It may exceptionally constitute the main disability of the disease. Sensations of numbness and tingling are experienced by some patients.

Vaso-motor phenomena are sometimes present. These consist of the so-called "dead fingers," passive congestion of the hand and arm, as shown by turgescence of the parts, local sweatings, and dryness of the skin.

Patients suffering from writers' cramp exhibit psychical symptoms. These are usually mental depression and a general nervous state of mind and body. They probably are the result of the disability occasioned by the disease, for the patient is fully aware of the loss of his former means of gaining a livelihood.

There have been differences of opinion as to the electrical reactions of the affected muscles. In true uncomplicated cases these are normal. When alterations are observed, it is more than probable that neuritis is present.

**Varieties.**—Four varieties of writers' cramp have been described. They are based on symptomatic differences only. They are: (1) The spastic; (2) the neuralgic; (3) the tremulous; and (4) the paralytic. The *spastic* variety is the one most frequently observed, and is characterized mainly by the tonic spasm of the muscles of prehension already referred to. The *neuralgic* variety closely simulates the spastic cases; but has, in addition, severe neuralgic pains. The *tremulous* variety is of rare occurrence; tremor takes place during the act of writing, but is absent during rest and while the patient is performing other movements. The *paralytic* variety is generally believed to depend upon a co-existent neuritis.

**Diagnosis.**—There is but little danger of confounding a well-developed case of writers' cramp with any other disease. In the initial stages of many organic diseases of the nervous system, as a slowly progressive hemiplegia, paralysis agitans, disseminated sclerosis, multiple neuritis, etc., the mistake of regarding the case as one of writers' cramp may be

made. This mistake is rendered likely because clumsiness in handling the pen is the first symptom of this disease which will attract the attention if the patient follows a clerical pursuit. Careful attention to the history of the case, and the presence of symptoms referred to other portions of the body than the writing hand, will save from error. Then, too, in writers' cramp the disability is observed only during the act of writing. All other complicated movements can be performed with the patient's wonted facility.

Imaginary disease may simulate writers' cramp. The differentiation is made by the history of the case, and by careful watching of the patient while he writes. In the simulated affection the symptoms are subjective and irregular. In the genuine, the spasm of the fingers will be observed.

**Prognosis.**—This is unfavorable, very few confirmed cases of writers' cramp being cured. Even though the patient educate the left hand to do the work of the right, that member will become affected in a comparatively short time in nearly three-fourths of the cases. This fact is taken as strong evidence of the central nature of writers' cramp. Cases in which much of the disability is dependent upon neuritis, offer a far better prognosis than do typical ones. Absolute rest of the parts for a prolonged term of years is not always by any means successful.

**Treatment.**—The most important element in the treatment of writers' cramp is absolute rest of the hand. It must desist from all writing. Many forms of apparatus designed to bring relief to the affected muscles by calling others into action, have been invented; but they are all open to the objection that they are not curative, and simply delay the time when total disability will result, unless complete rest is taken. At the very onset of the trouble, one month's rest will accomplish wonders, if not, indeed, cure; while if the case be permitted to proceed for a year or more, a rest of months will accomplish very little.

The patient should write with a soft quill or gold pen. His pen-holder should be thick and made of cork.

If writing must be persisted in, the patient should then learn to write with the left hand. It is true, that in many instances this member follows the fate of its fellow; but even then the patient is no worse off than he was before.

Those whose writing is not in books, should learn to use the type-writer.

Anxiety and depreciation of the general health standard serve to intensify writers' cramp. For this reason, constitutional measures are often of considerable value, in the early stages of the disease. The patient should be given good food and plenty of fresh air.

Physiological medication accomplishes nothing in the occupation neuroses.

Hughes suggests the use of *arnica* as a curative remedy. Cases of piano-players' cramp and writers' cramp have been reported cured with *gelsemium*.

Electricity is of considerable value in some cases. Galvanism is the preferable current. Beard and Rockwell recommend both central and peripheral treatment. "Galvanization of the upper portion of the cord and of the median and radial nerves, spinal cord, plexus and nerve currents, and faradization of the affected muscles and of their antagonists, may be tried; and when anæsthesia exists, the wire-brush."

Massage, systematically applied, has cured some cases in the hands of skilful manipulators. A writing-master from Frankfort, Germany, by the name of Julius Wolff, created quite a stir several years ago by his claims effecting wonderful results in the treatment of this affection. He kept his method for the most part secret; at least, all his descriptions, as published in the medical journals at my disposal, are so indefinite as to be valueless for practical purposes. From all that can be learned, his method does not differ from that practised by Dr. M. Roth for a number of years past, which consists of systematic manipulation of the entire upper extremity, after which the patient performs voluntary movements of nearly every conceivable kind with the affected fingers. Next, the operator proceeds to make passive movements of the parts, the patient resisting him.



## NEURASTHENIA.

**Synonym.**—Nervous exhaustion.

**Definition.**—A condition characterized by weakness or perversion of nervous function, arising independently of organic disease, or from quite a variety of causes.

Whether or not neurasthenia should be accorded the dignity of classification as a clinical entity has been argued pro and con by eminent authorities arrayed on either side of the question. Certainly the mere naming of a series of clinical phenomena in any given case of neurasthenia gives an inadequate idea of the illness, for in no other condition or sets of conditions are so many factors necessary before the case in point can be fully understood. The causes at work do not explain as much as they do when other disorders are the subject of investigation, for the individual peculiarities of the patient oftentimes decide in just what direction the "nervous breakdown" will take place. In no other condition are hereditary factors, manner of living, early training, etc., so important. Pathologically, but little can be said of the condition. Autopsies on the few cases of persons dying with nervous exhaustion have given absolutely negative evidence. From a practical standpoint, we must recognize neurasthenia as having a well-defined individuality, for there is no other designation by which the cases of this character can be indicated. At the same time one must take care lest he use the name as a mere "waste basket," into which he casts all cases in which, through lack of skill or otherwise, he fails to find physical evidence of organic changes. As with hysteria, neurasthenic symptoms appear in the course of the last-named conditions; indeed, they are the symptoms for which the patient seeks the relief. Neurasthenia occurring under such circumstances is merely secondary, and must not be regarded as the actual disease present.

Elaborate attempts to divide cases of neurasthenia into numerous classes have been made. Thus we have a degenerative neurasthenia occurring in the young, dependent upon a nervous system hereditarily deficient; malarial, syphilitic, and post-influenzal neurasthenia, names suggestive of the origin of the trouble; certain local neurasthenias as sexual and gastric varieties, with symptoms directed to perversion of functions in these organs; cerebral, spinal and general neurasthenias, according as the brain, spinal cord, or general nervous system, is supposed to be at fault. There may be some convenience arising from the use of these terms under certain circumstances, but as a rule they are

of limited value, and can be disregarded. The qualifying titles "malarial" and "syphilitic" are certainly superfluous.

**Etiology.**—Constitutional causes are exceedingly important as predisposing to neurasthenia. Some persons exhibit a remarkable capacity for work or activity in certain directions, and no amount of use or abuse of the nervous system seems to affect them. Their health capital seems unlimited; waste is rapidly repaired, and their health standard remains as high as ever. On the other hand, we encounter individuals presenting just the reverse state of affairs. They are nervously deficient by birth, so that they are early exhausted by labors that are the merest child's play to the ordinary individual.

The exciting causes of neurasthenia are those tending to mental and physical depreciation generally. Common among these stands overwork. This, it has been said, can do no harm in a strong healthy individual. Such a statement may be true, did we deal solely with ideal constitutions. But this we do not do. When overwork is combined with worry and anxiety, or with excesses in one or another direction, then an exhaustion of nerve function ensues. Of the local causes or excesses, none is so prolific of this condition than is sexual excess, especially if indulged in some unnatural manner. It is extremely doubtful if continence ever gives rise to the condition. Living under unnatural sexual condition may do so, however. To illustrate, I might mention a case already referred to by me in another connection, that of a minister ignorant of sexual physiology, who undertook to abstain from intercourse, but still occupied the same bed with his wife, to whom he was devotedly attached. Sexual excitement was the natural consequence. His nights were nights of self-restraint. The efforts to stifle natural feelings were, of course, exhausting. Finally emissions occurred regularly, and their advent became a source of alarm, and he grew morbid. Proper sexual hygiene cured him.

Not every case succeeding unnatural sexual gratification is the direct result of the pernicious habit. So very common are masturbation and kindred evils that the whole human race would be neurasthenic were it the sure and sole cause of nervous exhaustion. It is a matter of common observation that many cases arise in persons who had not practised the habit to any great extent. In them the trouble seems to have arisen either from forfeiture of self-respect because of moral weakness, or from morbid mental conditions excited by reading quack literature. Some have contended that masturbation is no more prolific a cause of disease than is excessive sexual intercourse. Here lies an error. The excitement attending the former act is not a natural one, and the expenditure of "nerve force," if the reader will allow the expression, is greater; then too, masturbation is generally practised during prepubic or the adolescent period of life, times during which the system needs

every available resource for the making of the perfect man. In the absence of hereditary predisposition or morbid mental inclinations, nervous exhaustion following upon sexual excesses of any kind, may reasonably be expected to make a prompt recovery on abandoning the bad habit.

Some cases arise from grief, unrequited love, and other emotional causes; and some from religious doubts.

**Symptomatology.**—Neurasthenia resembles hysteria very closely in presenting a wide range of symptoms, so great indeed as to lead some to consider it the male counterpart of that disease. Early in the course of the condition there appears a mental and physical weakness. The patient finds himself unable to perform tasks which were once with him trifling work; or he finds it difficult to concentrate his mind on his business or studies. The mental condition changes. He becomes despondent or low-spirited, and even morbid at times. Should the patient be a woman, she displays an unnatural emotional temperament. The physical being also suffers, as exhibited in gradual emaciation and anæmia. Sometimes the condition first shows itself in a peculiar irritability. Trifling affairs occasion great annoyance. The patient often loses confidence in himself. He seems to have lost all power of making an effort to go about his usual duties. It may even happen that the condition of exhaustion is displayed when no effort of any kind is being made. Sometimes the patient's morbid fears take one definite direction, the natural result being a condition not far removed from a monomania.

In the majority of cases sleep is disturbed. There occurs either insomnia, or fitful sleep disturbed by dreams. In the beginning the wakefulness is more particularly in the early morning hours, at which time, too, mental depression is apt to be most marked. Other cases, instead of being wakeful, display an abnormal drowsiness.

Vertigo is not infrequently more or less troublesome. Actual headache is not often complained of. Various uncomfortable cephalic sensations, on the other hand, are frequently experienced. Prominent among these is a peculiar sense of distress or pressure, referred most frequently to the top of the head. Sometimes it takes the form of burning in the occiput extending down the spine.

Of the special senses, sight suffers most. The patient experiences asthenopic symptoms immediately on beginning to read. These cases often go the rounds of the oculists, receiving many different corrections, never obtaining relief until the constitutional condition has been bettered. The pupils display an unnatural mobility. Sometimes they exhibit the reverse of the Argyll-Robertson pupil, *i.e.*, they react with abnormal readiness to light, and but sluggishly to accommodative efforts. The peripheral portions of the retina generally tire first, so that concentric limitation of the visual fields is discoverable by the



perimeter. The chief disturbances of audition are found in tinnitus, and a morbid sensitiveness to sounds. General sensory symptoms are rarely absent. The most common of these are diffused pains, generally referred to by the sufferer as "neuralgia." Sometimes these are localized, as in the case of intercostal neuralgia. Backache is just as common. It may be experienced in any portion of the spine. Very often it is felt in the nape of the neck. Paræsthesiæ exist in great variety, and form the foundation of many absurd fears of impending bodily disaster. Formication and numbness in one hand and arm, or in one lateral half of the body, awaken fears of impending paralysis. Sensations of heat and burning, distress in the epigastrium, and oppression in the chest, occasion much alarm.

Vaso-motor symptoms are not wanting. Morbid sweatings and blushings, general and local, are sometimes prominent, indeed the sole features of the case. Especially noticeable is sweating of the hands, which are often cold and clammy. The disease impresses itself on the heart, as exhibited by palpitation and disturbance of the cardiac rhythm. Respiration is often affected, there being especially deficient expansion of the chest.

The sexual functions are practically always diminished. Erections are defective, and sexual desire is lessened. Emission is often abnormal, being early or delayed. Sometimes a sexual irritability is the main source of suffering. The slightest excitement causes erections, followed promptly by seminal discharge. Locally, the genitals are found cold and relaxed. The sexual symptoms are nearly always the occasion of much concern on the part of the patient, he dreading impotence, above all things, especially when the trouble has been brought on by excessive indulgence, natural and otherwise. Phosphatic discharge is interpreted by him as loss of semen; or he is apt to imagine that the semen presents abnormal appearances.

The stomach suffers, as shown by gastralgia, loss of appetite, and uncomfortable digestion. Marked pulsation in the epigastrium is very often observed. Flatulence after eating is common. Nausea even may be present. The functions of the liver are disturbed. The bowels are constipated.

Tremor is sometimes a marked symptom.

A very common form of neurasthenia is that observed especially in women during the climacteric period. The characteristic symptoms of this variety consists of cold chills and hot flashes, and numbness and formication in various portions of the body.

The deep and superficial reflexes are nearly always increased.

**Diagnosis.**—In the majority of cases, this is an easy matter, there being but little difficulty is distinguishing neurasthenia from ataxia, brain tumor, and other organic diseases of the nervous system, gastric

catarrh, "liver trouble," heart diseases, etc. Careful physical investigations serve to decide the question at issue. To differentiate nervous exhaustion from hypochondriasis, certain insanities, sexual perversions, and hysteria, is not so easy, and may even prove impossible.

**Prognosis.**—This is nearly always favorable when the cause of the trouble can be discovered and removed. It may happen that by reason of long continuance of the disease, secondary conditions of serious nature develop.

**Treatment.**—The most efficient measures for the cure of nervous exhaustion come within the domain of hygiene. The cause of the trouble must be discovered and removed. When mental or physical overwork is the cause, the patient must take a rest. Having recovered, he should see to it that his nervous system is not again overtaxed. Along with the rest a change of scene is invaluable. Some patients thrive best when sent into the wilderness to camp out. Others need to go to different health resorts. Still others find relief in an ocean voyage. In prescribing these changes in the life of the patient, the physician must remember that rest constitutes a diversion of mental and physical activity from the usual channels. The student will find it best to lay aside his books and indulge in out-of-door recreation, or in mild cases, simply change the character of his reading. The business man may take up "hobbies," to distract his mind from business worries. The athlete must take physical rest. The character of the rest to be enjoined then must vary according to the patient's previous mode of life and the character of his symptoms.

As to diet, this should be as nutritious as it is possible to make it. In the absence of lithæmia or renal disease or other contraindications, it should be highly nitrogenous. Animal and fatty foods are the best for the neurotic. They should, moreover, be partaken of as freely as digestion will permit. Very often it is advisable to have the patient fed something at short intervals, even if he takes nothing more than a glass of milk. The full diet necessary for the cure of these cases can rarely be reached at once, as the patients are firmly impressed with the idea of possessing bad digestive powers. Directing physical rest, it will be found that digestion improves, and with it the quantity of food administered should be increased.

Rest in bed is very important in many cases. It is rarely, however, that one is required to put the patient in bed for a prolonged period. The average case needs rest after meals, especially after dinner, when a nap of one hour should be made the rule. Other cases should be made to take breakfast in bed.

Cases with hysterical or hypochondriacal tendencies require isolation, generally in the shape of sending away from home and relatives.

Massage is often an important adjuvant.

Hydro-therapy furnishes us some valuable assistance in the treatment of neurasthenia. The cold spinal douche immediately on awakening in the morning is highly invigorating. The patient should be directed to run a few inches of hot water into the bathtub. He then sits on a board hung across the tub, while water from the cold water spigot is made to pour down his spine from the nape of the neck for fifteen or thirty seconds. He then rubs himself briskly with a coarse towel and dresses or returns to bed, according to the requirements of his case.

Among remedies, *strychnia sulph.* is the most useful. It may be given in doses ranging from one-hundredth to one-thirtieth of a grain three times daily. When anæmia is present, it may be judiciously combined with iron.

*Picric acid* is also a very valuable remedy. It has headache brought on or aggravated by the slightest attempt at using the mind. It is generally situated in either the forehead or occiput. When in the latter situation, it extends down the spine. There are more or less constant tired and heavy feelings. The sensations of heat, so common in many cases of neurasthenia, are characteristic of picric acid. Various visceral sensations are often present in the cases calling for this remedy. When spinal symptoms predominate, the *picrate of zinc* 2x is a better remedy.

*Phosphoric acid*, though generally used in cases of sexual neurasthenia, is equally well adapted to cases arising from overwork. Both brain and spine are exhausted. A peculiar vertigo is sometimes complained of, namely, on lying down there is a feeling as if the head was going higher than the head. Seminal emissions are frequent. The genitals are cold and relaxed. Pains are absent. Profound mental and bodily weakness is the characteristic indication.

Very many other remedies are useful in neurasthenia, among which may be mentioned *argentum nitricum*, *nux vomica*, *phosphorus*, *silicea*, *oxalic acid*, *actea racemosa*, *ambra grisea*, *sepia*, *platina* and *sulphur*. In the selection of a remedy the greatest judgment should be exercised in observing symptoms, lest the physician increase the patient's morbid tendencies thereby. Many very peculiar symptoms must be regarded as valueless from a therapeutic standpoint, being the product of the patient's imagination, and often curable by a good talk or a few positive statements.



## EPILEPSY.

Epilepsy is a chronic disorder of the nervous system, characterized clinically by recurrent convulsions of a type to be hereafter described, and associated with loss of consciousness; in other cases the paroxysms consist solely of loss of consciousness, attended or not by various sensory or mental phenomena. In all cases the attacks or spells are sudden in onset, and of comparatively short duration. During the intervals of the seizures, the patient is apparently well; at least he presents no symptoms indicative of the epileptic state.

It is now becoming a well-recognized fact that epilepsy is but a generic term intended to include a variety of paroxysmal convulsive disorders. Some of these are known to be definite organic diseases; others, despite most carefully conducted pathological research, fail to show any pathological lesion. These latter are in the present state of our knowledge regarded as cases of idiopathic epilepsy, and it is to them that this chapter refers exclusively.

Even idiopathic epilepsy has been subdivided into varieties. Thus we read of *epilepsia procursiva*, *epilepsia larvata*, *epilepsia saltatoria*, *epilepsia gravior* and *epilepsia mitior*. Many other varieties have been mentioned in literature. Space will not permit even a mention of them here. Reference has been made to those above, not to define or describe them, but merely to acquaint the student with their existence, and to say that they find their existence solely on symptomatic and not, so far as we at present know, on pathological differences.

*Epilepsia gravior* and *mitior* have long been mentioned in text-books, and are synonymous with *grand mal* and *petit mal*, or severe and mild seizures, respectively.

**Etiology.**—The etiology of epilepsy is enshrouded in obscurity; practically all the data bearing thereon are of a general character. We may divide the causes into predisposing and exciting, or remote and immediate, the former being decidedly the most important. In many cases an exciting or immediate cause is absent.

There is a well-defined relation between epilepsy and age. While at no time of life is humanity exempt from it, the majority of cases set in between the tenth and twentieth years, and most of these at about the age of puberty. Probably 12 per cent. find their onset in infancy, that is prior to three years of age. Fully 75 per cent. occur during the developmental period of life, that is prior to the twentieth year. Epilepsy has even been known to attack patients as old as seventy. In many

of these, it is probably true that the convulsive seizures are the result of atheromatous changes in the intracranial vessels, and so cannot be looked upon as due to *idiopathic* epilepsy.

Epilepsy attacks the sexes in nearly equal proportion; the number of cases in females is slightly in excess of that in males.

*Hereditary predisposition* is undoubtedly a powerful etiological factor in a respectable minority of cases, variously estimated by different authors at from 10 to 40 per cent. Epilepsy is, however, very rarely transmitted from parent to child as such. Usually a neurotic constitution in the parent, as evidenced by insanity, neurasthenia, neuralgia, etc., is transmitted to the child as epilepsy. Likewise, epilepsy in a parent may show itself in a child as one of the neuroses above mentioned.

*Hereditary syphilis* has been accused of being a prolific cause of epilepsy, and with apparent good reason, for, in many cases, a history of syphilis in the parents is obtainable. Still we must not lose sight of the fact that where syphilis produces disease of the nervous system, the lesion is not apt to be of the mysterious nature of that exciting epilepsy. The majority of cases of syphilitic epilepsy, so-called, will be found, on close study, capable of being classified with organic cerebral disorders.

*Alcoholism* in the parent is unquestionably productive of epilepsy in the offspring, and for two reasons. In some of these cases the alcoholic craving is but the expression of a neurosis which is transmitted; and this transmission is aided by the faulty nutrition which the long-continued alcoholic addiction must occasion. Cases have occurred in which the alleged cause was the production of conception by a coitus during which the father was intoxicated.

There is no doubt also that certain diathetic diseases, as tuberculosis, rachitis and gout, exert an etiological influence in some instances.

So much for the predisposing causes of epilepsy. The *exciting causes* of the disorder are far more numerous; at least, what is alleged to be "exciting causes," are frequently observed. A frequent cause is to be found in the acquirement of the epileptic habit in early infancy. While there can be no question concerning the teaching that the vast majority of cases of eclampsia in children of less than three years of age are either reflex in origin or symptomatic of some acute disorder, still sight must not be lost of the fact that some of these are epileptic *ab initio*, and that in other cases the frequent recurrence of reflex infantile convulsions finally establishes a convulsive habit, which finds its expression in later life as a full-fledged epilepsy. Careful investigation of many cases of epilepsy will show that the patients suffered from infantile eclampsia.

*Emotional influences* have been credited with producing the first convulsion. Usually the emotion blamed is one of a depressing or unpleasant character, as grief or fright. It is not necessary that the epilepsy

follow immediately upon the receipt of the mental shock. On the contrary, it has been said that the longer the interval (within reasonable bounds, of course) between the exciting emotion and the ensuing convulsion, the more likely is the latter to be the beginning of epilepsy. Lest some may doubt the influence of a simple mental shock in producing such a serious disease, it is well to state that such a cause, acting but the once, has been known to produce cerebral degeneration, ending in death. Of course, we may meet with patients in whom the epileptic habit is developing, and who are just waiting for an excitant to bring forth the first seizure. In them the attack may follow immediately upon the so-called exciting cause. These patients may be said to be in such a condition that any unusual influence may become operative in this respect.

*Injuries* are universally recognized as frequently the cause of epilepsy. The character of the head injury does not seem to be of especial importance. Ofttimes it is a depressed cranial fracture. Just as often it is an injury without perceptible involvement of the scalp or skull. It is safe to say, however, that epilepsy is decidedly less liable to follow cases of head injury properly treated. The length of the interval between the reception of the injury and the first epileptic convulsion presents the greatest variation in different cases. It is rarely, if ever, very short, and usually it is sufficiently long for secondary sclerotic changes to have taken place in the brain, and become the cause of the discharging lesion.

Certain acute diseases, as scarlatina, measles, typhoid fever, and inflammatory rheumatism, are alleged causes in some instances. The exact relations of these diseases to epilepsy is probably not understood.

Many times digestive derangements are credited with having brought on the first attack. The usual history is that of a convulsion having occurred shortly after excessive indulgence in indigestible food. There is a strong tendency on the part of physicians to diagnose such cases as reflex convulsions, thus committing, as I believe, a serious error, as will be shown presently. It is essential that the role of the stomach in exciting and perpetuating epileptic attacks be recognized, for herein is found the most important element in the treatment of the disease. Yet we must observe care lest we fly to another extreme, and make gastric disorder the *fons et origo* of epilepsy.

There is but little reliable evidence to show that epilepsy is produced by sexual excesses or unnatural sexual practices. The true relation between the two conditions is probably that the mental degradation so frequently associated with epilepsy is the foundation for the disgusting sensuality that is itself credited with being the etiological factor.

Menstrual derangements can but rarely be said to be productive



of epilepsy. The majority of cases of convulsive seizures occurring at the menstrual periods are of the hysterical type. Very rarely an epilepsy finds its exciting cause in the local derangement attendant upon abnormal menstruation. More frequently, the developmental changes which are going on at the age of puberty, and which in themselves are the cause of the menstrual difficulty, are the real excitants of the epilepsy.

Other exciting causes of epilepsy, causes to be found in exceptional instances, are alcoholism, lead poisoning, renal disease, tobacco poisoning, pregnancy, and excessive mental application.

In some cases certain *local irritations* seem to be productive of seizures, as refractive errors, decayed teeth, contracted and irritable prepuce, foreign bodies in the ears and nose, etc., too numerous to mention. The practical importance of their recognition as causes of reflex irritation is not that by their removal epilepsy is safely and immediately cured, but rather by their removal the chances of a complete recovery in any given case of epilepsy are greatly enhanced.

The etiology of epilepsy has been considered at this great length because the proper understanding of even the few facts at present in our possession is of the greatest importance as bearing on therapeutical questions.

**Symptomatology.**—The usual history of a case of epilepsy unmodified by treatment is as follows: Within a short period after an alleged exciting cause a convulsion occurs. This convulsion lasts probably from three to five minutes, is followed by a sleep more or less prolonged, from which the patient awakens. In a short time, that is, within a few hours, the previous state of good health is resumed, and continues until the next seizure appears. The convulsions recur with greater and greater frequency until in some cases they may be repeated a number of times daily. Close questioning often shows that the patient is also subject to attacks of momentary unconsciousness, attacks of *petit mal*. As the disease increases in severity, mental enfeeblement is noted, and the natural progress of this is often to dementia.

The typical complete epileptic convulsion consists of several stages as follows: The aura, the cry, the stage of tonic spasm, the stage of clonic spasm, and the stage of stupor. During all of these stages, with the exception of the first, the patient is unconscious. One or more of these may be absent, and yet the seizures be none the less epileptic. The aura is, in my experience, to be found in only a minority of cases. We may define this term as any subjective sensation, motor phenomenon, or psychical manifestation immediately preceding, so as to almost form part of, the convulsive attack. The term aura originated from the fact that in the majority of cases in which it was present, it consisted of a sensation as of a draught of cold air passing over the affected parts. It must

be understood that the epileptic aura may consist of any symptom. In each individual case, the aura is nearly always the same in all seizures. The most frequent variety met with is that of a vague indefinite sensation, sometimes described as a sensation of heat, or of a draught of air, starting from the epigastrium and travelling upwards to the head. When it reaches the latter, the patient becomes unconscious. In other cases the sensation starts in an extremity and proceeds upwards. Sometimes the aura consists of a special motion, or of a peculiar mental expression.

Immediately following the aura, and coincident with each other we have the epileptic cry and the stage of tonic spasm. The cry is given forth at the beginning of the tonic stage, at the moment the patient lapses into unconsciousness. The cry is supposed to be due to the sudden tonic spasm of the muscles of respiration forcing air through the larynx, which is also in a condition of tonic spasm. The cry is by no means an essential feature of the disease.

The tonic spasm comes on so suddenly that the patient falls before he has an opportunity to reach a place of safety. The body is held rigid. The head and eyes are turned towards one side or the other, most frequently the right. The jaws are fixed. The eyes are open and staring. The face, which at first was pale, becomes dark and congested. The extremities are drawn into various positions by the most strongly contracted muscles. Usually the legs are extended, and the arms flexed at the elbows, and the hands clenched. The rigidity of the muscles of respiration gradually brings on a cyanotic condition. During the tonic stage the conjunctivæ are insensitive, and the pupils become widely dilated.

Gradually the tetanic rigidity of the body may be felt to give way, and clonic movements of the affected muscles are to be noticed. These gradually increase in intensity until the stage of clonic spasm is fully developed. These movements are of a decidedly shock-like character. Involving the muscles of the thorax, saliva is forcibly and intermittently ejected from the mouth by the air, and so becomes frothy. Gradually the clonic stage disappears, not by the individual movements of which it is composed becoming less strong, but by their frequency diminishing. Finally they cease, and the convulsion is at an end.

Then ensues the stage of stupor, from which the patient may sometimes be aroused, though with difficulty. From this he awakens occasionally feeling as well as ever, though more frequently exhausted.

The tonic stage usually lasts less than one minute; the clonic stage four or five minutes; the stupor is of indefinite duration.

It sometimes happens that during the spasm there is involuntary discharge of urine and fæces. This unfortunate occurrence is met with in but a minority of the cases; but in those cases in which it is a symptom, it is a concomitant of nearly every seizure.

Usually the muscles of the two sides of the body are unequally affected.

The temperature during the convulsion is almost always above normal, oftentimes two degrees. The normal is reached about two hours after the cessation of the attack.

The knee-jerks are, in violent cases, entirely absent or obtained with difficulty immediately after the seizures. They remain so for a period ranging from ten to thirty minutes. They will then appear in some cases exaggerated. The knowledge of the condition of the knee-jerks after epileptic attack is of importance as an aid to detect malingerers. During the intervals of apparent health between the convulsions, the knee-jerks are normal.

The condition of the urine of epileptics has many times been made the subject of study. Urea excretion following seizures is usually excessive. Albuminuria and glycosuria are but rarely noted, and must be regarded as complications of the disease. Recently, attention has been called to excess in the ethereal sulphates as indicative of intestinal disorder preceding the seizures.

Careful investigation of the eyes by unprejudiced experts shows nothing abnormal with these organs in epileptics.

The time at which attacks come on is often interesting. In some cases, they appear only during the day; in others only at night, and at a particular hour thereof; in still others they occur during both day and night. In women, they are not infrequently aggravated before or during the menstrual periods.

It must not be forgotten that single fits may be epileptic. In such cases the disease is not deeply rooted, and the tendency to recur is never called into activity.

**Petit mal; Epilepsia mitior.**—Practically speaking, attack of *petit mal* consists of but the aura plus unconsciousness. The usual history of a case is a "spell" in which the patient, without warning, gives a staring expression, and in less time than it takes to tell about it, is himself again. He may stop in the midst of some work, which he resumes at once on returning to his normal condition. The following characteristics of *petit mal* have been mentioned by Gowers: Vertigo, simple jerks, visual sensation or loss, mental states, unilateral peripheral sensations or spasms, epigastric sensation, sudden tremor, pain or other sensations in the head, choking sensation, sudden scream, olfactory or cardiac sensations, sensation in eyeball, sudden dyspnoea, and general indescribable sensations. With all of these the important characteristic that makes them diagnostic is their sudden onset, their momentary duration, and the apparent good health of the patient both before and after the "spell."

In many cases, the minor attacks are alone noted. More frequently, however, they are found in association with the convulsive seizures.



A very practical question is that of mental disturbance in epileptics. One undoubtedly meets with cases of epilepsy in which the highest type of mental development is found. Such cases are exceptional. As the disease progresses unchecked by treatment, there ensues a blunting of the mental faculties, loss of memory being especially prominent. Sometimes complete dementia supervenes.

There are other mental phenomena attendant upon epilepsy that are exceedingly interesting, not only to the physician, but to the medico-legal expert. Thus we note certain psychoses with more or less loss of consciousness; others not so accompanied. Sometimes when consciousness has returned, the patient has hallucinations of grotesque purport, and these may keep the patient in constant terror during their continuance. Sometimes the fits are followed by automatic actions; sometimes by a delirious frenzy in which the patient is destructive, if not actually murderous. Usually these physical phenomena are characteristic of the cases in which they occur, and are readily accorded their proper value in the understanding of the case.

Imbecility and idiocy, associated with epilepsy, are but expressions of organic cerebral changes, either acquired or congenital.

**Diagnosis.**—It is important to differentiate epilepsy from reflex convulsions, uræmic convulsions, convulsions from organic brain disease, hysteroid seizures, syncope, vertigo, malingering and symptomatic epilepsy. Of these, the most important for differentiation, because the most frequently thought of, are reflex convulsions. Concerning this subject the wildest and most absurd errors are rampant among the profession, and these are nurtured by the one-sided view of medicine taken by partially educated, but none the less enthusiastic, specialists.

It is a good rule to follow in the case of a convulsion occurring for the first time, after the third year of life, to consider the probability of its being epileptic as very strong indeed. Convulsions prior to that age are, in by far the majority of cases, either reflex in origin or symptomatic of organic disease. Reflex convulsions nearly always give a history which points out the seat of the causative irritation. In the case of a cicatrix, we should expect to find it sensitive to pressure, etc. When the attacks are dependent upon ovarian or uterine difficulty, symptoms in those organs establish the relation of cause and effect indubitably. So far as the character of the attacks is concerned, reflex convulsions generally simulate the epileptic so closely as to generally make a differentiation by their aid almost impossible. The reflex seizures are not, perhaps, as sudden in their onset, and in them the disturbance of consciousness is not as profound as in epilepsy. Their duration is generally longer. Epileptic seizures are usually over in five or ten minutes; reflex convulsions may be of indefinite duration. When one reflex convulsion has occurred, it is very apt to be followed shortly by others in

rapid succession. In the case of epilepsy, the patient is apparently well after the attack, and the second convulsion does not follow for some time, oftentimes for nearly a year. Having diagnosed reflex convulsions, it is always a wise plan to doubt one's diagnosis, and care for the patient as if he might possibly become epileptic.

The advanced stages of interstitial nephritis are not infrequently accompanied by recurrent convulsions, the character of which does not differ from those of epilepsy. The diagnosis is to be made by the age of the patient (most of these uræmic cases occurring after middle life), the condition of the pupils, the presence of albuminuria, a low, urinary specific gravity, deficient elimination of urea, hyaline casts, high arterial tension, and the duration of the seizures. In rare instances, the convulsions of contracted kidney may be purely local in distribution, and simulate Jacksonian epilepsy in character. Care is here required to differentiate them from convulsions of organic brain disease.

The typical convulsions of organic brain disease are of the Jacksonian type. Paralysis ensues in the previously convulsed parts. In the majority of instances, however, the lesion is so situated as to produce a general convulsion indistinguishable in character from the epileptic. The attacks in these cases are much longer than those of epilepsy, the patient is usually advanced in years, consciousness is not necessarily lost, and between the seizures, there is a history of headache, vomiting, optic neuritis, vertigo, and other symptoms of cerebral disease.

Epileptiform attacks occur at the beginning of many acute diseases, and as death comes on from hæmorrhage. The diagnosis of such cases is self-evident.

For the differentiation of hysteroid and epileptic seizures, there is no better guide than the table given by Gowers, and this is as follows:

	EPILEPTIC.	HYSTEROID.
Apparent cause.	Absent.	Emotional disturbance.
Warning.	Any, but especially unilateral, or epigastric aura.	Palpitation, malaise, choking, bilateral foot aura.
Onset.	Commonly sudden.	Often gradual.
Scream.	At onset.	During course.
Convulsion.	Rigidity, followed by "jerking," rarely rigidity alone.	Rigidity, or "struggling," throwing limbs and head about.
Biting.	Tongue.	Lips, hands, and more often other people and things.
Micturition.	Frequent.	Never.
Defecation.	Occasional.	Never.
Duration.	A few minutes.	Often half an hour, or several hours.
Restraint.	To prevent accident.	To control violence.
Termination.	Spontaneous.	Spontaneous, or artificial (water, etc.).

Cases of hysteroid seizures will be occasionally noted in which the convulsive movements are of an epileptiform nature. We may also meet with cases in which hysteria is added to epilepsy or other epileptiform disorder. The history of the case and the collateral symptoms must then guide us to a conclusion.

The simulation of epilepsy is recognized by the condition of the pupils and the condition of the deep reflexes. The observer must not forget that excessive voluntary movements, such as must be made in order to simulate an epileptic attack, can produce dilated pupils. Such pupils will contract, however, to the stimulus of light. Another plan for the detection of simulated attacks is to blow snuff in the nostrils, which will then cause sneezing. It will not have this effect if the attack be genuine.

Syncope is to be distinguished in that its attacks generally occur in the constitutionally weak, or from some well-defined cause. They are more gradual in their onset, and are of comparatively long duration.

The same data enable us to differentiate vertigo from vertiginous epilepsy.

Nocturnal epilepsy sometimes escapes recognition for years. Trousseau has given us as data which should lead us to suspect its existence: Incontinence of urine at night; awakening in the morning with headache; difficulty in speech, owing to having bitten the tongue during sleep, and purpuric spots on the face and neck. None of these symptoms, singly, has diagnostic importance; but, collectively, they should awaken a strong suspicion of nocturnal epilepsy.

**Prognosis.**—At the best, the prospects of a radical cure in a given case of epilepsy are decidedly uncertain. Many cases are radically cured. Some cases recover spontaneously, as the result of proper hygiene. Other things being equal, cases offer the better prospects as they come under skilled observation the earlier, and the fewer the number of attacks. Cases in which attacks of *grand mal* and *petit mal* are both found, do not offer as good a prospect of radical cure as do others. Marked mental degradation makes the prognosis decidedly bad. The epileptic attacks rarely result in death; though the danger of suffocation from the patient burying his head in the pillow and similar accidents must be borne in mind. The status epilepticus is, however, dangerous to life, the majority of cases dying. Patients with epilepsy are not long-lived, generally dying comparatively early of some intercurrent disorder.

**Treatment.**—Many of the bad results in the treatment of epilepsy are undoubtedly due to a lack of knowledge as to what constitutes the disease. An experience that is by no means small, leads me to assert that the vast majority of cases, I might almost say all, are diagnosed in their incipency as reflex convulsions. This error is not unnatural when



one considers the stress that has been placed on the frequency of reflex convulsions by certain authorities. Specialists whose training in the domain of general medicine has been sadly deficient are likewise largely responsible for the promulgation of what, as applied to most cases of epilepsy, is a false theory. It is a good rule to regard every case of convulsion, *no matter how probably reflex*, as *possibly* epileptic. While one is generally pretty safe in deciding that convulsions not due to organic disease occurring prior to the third year of life are reflex in origin, he must not lose sight of the fact that their frequent repetition can give rise to the convulsive habit; and thus true epilepsy finds its origin. It is not a pleasant task to speak to a parent of the possibility of his child degenerating into an epileptic; and yet a strict regard for duty should lead us to give that warning, for I believe that such a warning will do much to prevent a probability or possibility from becoming a reality. That this warning is not an idle one will be shown if one but take the trouble to investigate the early history of epileptics. In more than half the cases, probably, a history of infantile eclampsia is obtainable.

And right here let me give a little piece of advice that should be adopted as a routine measure in managing every case of epilepsy: Direct the patient's attendant to keep a day-book. In this book should be recorded the number of convulsions, their characteristics, time of onset, duration, attendant circumstances, etc. All morbid phenomena occurring between seizures should likewise be carefully noted. Such a book has a two-fold purpose. In the first place, it affords us a certain amount of help in treatment of the case, not otherwise obtainable; and in the second place, it is invaluable as indicating most certainly the progress made. Certain hospitals treating large numbers of epileptics have specially prepared charts for indicating the number of convulsions. By means of this device one can see the course of the disease, so far as it relates to the number of the convulsions, at a single glance. Without some such record as the day-book or chart, any testimony as to improvement or cure is more or less unreliable.

When once epilepsy has been developed, the plan of treatment which must be followed must include careful observance of all the rules of personal hygiene, including regular habits and careful dieting, removal of all possible sources of irritation, and proper medication, and in suitable cases surgery.

Considering first the question of diet, we come to one of the most important items in the treatment of epilepsy. Some authors do not hesitate to make this the first in importance. Here we find authorities at variance, according as they take this or that pathological view of epilepsy. Seguin, Gowers and others taking the ground that the nervous system should be fed with the strongest food, as in neurasthenia, do not hesitate to strenuously advocate a diet into which meat enters largely

Haig, believing that epilepsy is a disease resulting from the uric acid diathesis, prohibits meats; others, while not agreeing with Haig as to the origin of epilepsy in uricæmia, consider the meat diet positively harmful as promoting a convulsive tendency. Clinical facts seem to favor the latter view, in my opinion. I think it wise, therefore, in the majority of cases, to keep epileptic patients on a mainly vegetable diet, allowing milk regularly, and poultry and fish on rare occasions. Even the administration of liquid preparations of meat, as soups, broths, peptones, and the like, has a bad effect on the epileptic. I have been assured by those in charge of institutions receiving epileptics that these cases are almost invariably made worse by such a diet.

Of far more importance than the general character of the diet is the quantity eaten, and the manner of taking it. Nothing is more harmful than overloading the stomach, especially with articles proverbially indigestible. Above all things the food must be slowly eaten. These are directions which will require the greatest care to enforce, for epileptics usually have ravenous appetites, and exhibit no judgment whatever in satisfying them.

It is needless to say that indigestible foods and mixtures, as pastry, mince pies, etc., etc., should be forever eschewed.

In cases in which the seizures are almost invariably nocturnal, the evening meal *must* be taken as early as possible, and should be of the lightest possible character.

In every case the relation of attacks to diet and times of eating should be carefully studied, and our advice should be governed accordingly.

Any disorder of the stomach must be carefully treated. In fact, it is of the highest importance to keep this organ in as good condition as possible.

In thin anæmic cases, cod-liver oil is a positive necessity.

Alcohol and alcoholic preparations are positively harmful.

Dancing, swinging and other amusements likely to influence the cerebral circulation deleteriously, must be forever forbidden. One patient, in whom I had been fortunate enough to keep the attacks away for over a year, had a relapse from using a swing.

One cannot lay down any hard and fast rules concerning occupation, general amusements, etc., as each case must be studied *per se*. In restricting the life of these patients one should not go to the extent of depriving them of amusements, etc., so completely as to make life a burden.

The general care and management of epileptic patients is a matter of unrecognized importance. Here I use the word care to include "discipline" as well. Parents lack firmness and wisdom oftentimes in the management of their own, and by injudicious "coddling" do much to render ineffective well-directed efforts of the physician at cure. Many times the best results can only be obtained away from home.

Sexual hygiene is an important matter. I entertain very serious doubts that sexual excesses ever caused epilepsy, though it is certain that they aggravate it if it once develops. Epileptics are unquestionably great masturbators, probably because the disease produces a mental degeneracy leading to that sexual vice. Masturbation, then, must be stopped. The nearer the patient comes to celibacy, the better will it be for him.

It is an important matter to keep epileptics from dangerous places.

The reflex origin of epilepsy is a fruitful source of discussion. Physician after physician can tell us of cases that he has cured by this or that minor surgical procedure; and yet the details are barely more complete than they should be for a well-told anecdote. It is singular that each enthusiast has his particular operation which will cure all cases. One man can tell us of countless cases cured by circumcision. Our friend, the oculist, may tell of cases cured by fitting for glasses; the aurist, of cures effected by attention to the ears. The neurologist, notwithstanding his careful attention to all possible sources of irritation, says that he cures his cases with the greatest difficulty, and in exceptional instances only. For awhile, the gynecic surgeons were oöphorectomizing the poor helpless epileptic. So far as my knowledge of medical literature goes, I know of no case in which a permanent cure has been effected by this operation other than in those to which I shall refer presently.

It is a wise plan to remove all possible irritation from the areas of distribution of cranial nerves. In keeping with this idea, teeth, eyes, ears and nose should be carefully examined and kept in order. Under no circumstances should the wild fads and fancies of the Stevens school of ophthalmologists receive sanction. Muscular anomalies may be found occasionally, and should, of course, receive *scientific* attention; but under no circumstances should the *wild resort to indiscriminate* tenotomy be advised or permitted.

Circumcision is wise as a preventive of masturbation. It is necessary when the redundancy is the cause of local irritation, or where there is an obstruction to the escape of urine. Circumcision is not an absolute preventive of self-abuse, as the habit is not uncommon among Jewish boys.

Many measures have been recommended for the abortion of the fits. Ligation of an extremity when the aura begins in that part has been practised with some success. In similar cases blisters encircling the arms have likewise done some good. It is an interesting as well as noteworthy fact in these cases that after the fit has been stopped at the site of the ligature or the blister several times, it acquires the habit of ending there in subsequent attacks, though there be no mechanical device there to prevent it. Of course, this relief is but temporary.

*Nitrite of amyl* has been used as a preventive of attacks, but it is beneficial in only a very small percentage of cases.



The use of salt and other agents during the spasm does no good. The fit must run its course.

The only treatment applicable to the convulsive stage is that which will protect the patient from injury.

The medicinal treatment of epilepsy, viewed from whatever standpoint we may, is not in a satisfactory condition. From our homœopathic remedies we do not secure any regular results, while the bromide of potassium is but a palliative.

As to *œnanthe*, *cicuta virosa*, *hydrocyanic acid* and other so-called specifics, I have never seen one satisfactory result, notwithstanding the large number of cases in which they have been administered. It is but just to say, however, that they were given mostly in dispensary cases, in which class one must rely almost exclusively upon drug treatment, for dispensary patients have neither the intelligence nor the disposition nor the means to pursue a proper hygienic course. In private practice my cases had all been previously treated over terms of years by physicians of both schools. Dr. W. M. Butler reports having cured several cases with hydrocyanic acid, but he gives no particulars as to the character of the cases or the time over which treatment was extended. The most successful medication seems to be that in which the aim is to keep the general health up to the highest standard. *Nux vomica*, *belladonna*, *pulsatilla*, *bryonia*, *cuprum*, *plumbum*, *hyoscyamus*, and like remedies are not only valuable, but necessary.

Much has been said of the tissue remedies. I have tried Schüssler's *kali mur.*, but without the slightest sign of success. *Silicea*, *calcareo carb.* and *sulphur* have done considerable good when systemic conditions indicated those remedies. *Argentum nitricum* in one case effected a cure that lasted two years, when there was a relapse. I know nothing of the subsequent history of the case.

In the case of failure to get epileptic seizures well under control within a reasonable time, there should be no question concerning the resort to bromides. Bromide of potassium has been abused to such an extent as to deter the timid from its use. It has been accused of producing a condition far worse than the original trouble for which it was given. Physicians of all schools unite in its condemnation. Why should it not do harm when used ignorantly or indiscriminately? And yet, if the drug is used with judgment, it is the greatest boon certain epileptics have. When it is suited to the case—that is to say when it is given in cases in which it causes a cessation of the convulsions—it most assuredly improves his mental faculties instead of depressing them. I have seen this over and over again. There need be no fear of giving large doses. One of the reasons for failure hitherto with most physicians is that the maximum dose given is but thirty grains daily. In the majority of cases such doses are inefficient, and, unless they control the fits, must show a

depressing influence on the mind. When, however, an efficient dosage is adopted—and by efficient I mean quantities sufficient to keep the fits away entirely—one will be astonished at the improvement in the patient's general condition.

One naturally views the effects of bromides in epilepsy according to his individual experience. I do not wish to convey the idea that the drug is without ill-effect in all cases, or that it is always curative or even palliative. On the contrary, I know this is not the case. But there are a large number of epileptic patients whose natural tendency is towards dementia. They are desperate cases, and they go from bad to worse. Most of them have organic cerebral disease as their anatomical substratum. No drug will do them one iota of good. Yet they have been dosed, not only with bromide, but with every drug conceivable. Quack medicines have been administered by the score, and bromides have been administered by the five cents' worth, without the sanction of a physician. Is it any wonder that such cases end in the asylum? At home their friends are often too lazy or too poor to give them the proper care, and this only adds to their miseries. Such cases are often met with as well in the families of those who are careful, but here good treatment does much to prevent the terminal dementia.

In dispensary practice one meets with a middle class of patients who follow advice perfectly so far as it relates to medicinal treatment, but who will not or cannot obey general directions. Such patients cannot expect to obtain the best results from carefully-given medical advice.

If one wishes to avoid bromism, and would carry the palliative treatment of epilepsy to its greatest efficiency, he must pay the greatest attention to the minutest details. The fact that epilepsy is best treated palliatively by bromides should not lead to routine carelessness.

The dose that will be efficient in any given case can only be decided by careful observation of that case. Repeated visits to the physician, I might almost say daily visits, are necessary to decide this point. The smallest dose that will suppress the seizures is the one to employ. I would not be satisfied with a partial suppression. I would aim at making the suppression absolute. Of course, this result can be obtained only in a minority of the cases; then one must be satisfied with the dose that produces the maximum effect without exciting bromism.

One of the unfortunate effects of bromide of potassium in its tendency to disorder digestion, an effect, too, that has more to do with the production of bromism than appears at first sight. We have two measures that obviate this to a great degree, namely, large dilution of the dose, and the administration of the drug in a feebly alkaline mineral water, as Vichy. These are highly important points, which must be observed in every case. Given thus, the medicine is much more rapidly absorbed. It should likewise be drunk slowly.

It is better to give the drug in as few doses as possible. I would never give it oftener than three times daily. With the majority of people it is a difficult, if not almost impossible matter, to get them to take medicine regularly over any great length of time. The longer the intervals between doses the more have we reason to expect regularity. As to the times at which the drug should be given, we must be governed by individual cases. One dose should be given as early in the day as possible; while another should be given at an hour about from four to six hours before the usual time at which the fits are likely to occur. Gowers recommends large doses at long intervals, thus, one drachm every day, two drachms every two days, three drachms every three days, etc. The longer the interval between the doses, the larger is the dose prescribed. He claims that in this way the maximum curative effects are obtained, with the least liability to bromism.

One can never, and I mean never most emphatically, entrust the administration of the drug to the patient himself. I never saw an epileptic who would take his medicine regularly unless he had some one to attend to it for him. This is true, notwithstanding the strongest incentives to recovery; it becomes more forcible when medicinal action has secured an all but permanent cessation of seizures. Epileptic patients are their own worst enemies.

Having once begun the administration of the bromides, it is necessary to keep them up steadily for years. It has been said that a case cannot be considered as cured until the fits have remained away for three years. I would err on the safe side, and say that one can never say when a case is cured. The fits may have remained away for five nay, even ten years, and yet a relapse may occur. I would insist upon continuous medication for a period never less than three years after the last seizure, and, if possible, I would keep the patient under the drug for five years. I do not know but that if I were an epileptic I should insist upon taking it all my life.

In female cases, in which the attacks seem to recur more frequently about the menstrual periods, it is advisable to give the drug in larger doses then than at other times. I should say here that the *aggravation* of attacks about the menses does not by any means signify a reflex origin of the disorder.

Bromic acne has been greatly feared. This deleterious effect of the drug should not occasion the slightest anxiety. It may be entirely obviated by the administration of Fowler's solution of arsenic. The bromic acne is not an index of the physiological effect of the drug. The readiness with which it is produced, depends largely upon peculiarities of skin structure.

Some patients are more susceptible to the unpleasant effects of bromide than others. While small doses exert a depressing influence in a



few, there are others who can take the most extravagant doses, without a symptom being produced thereby. Other things being equal, children seem to be able to withstand bromism better than adults. Small, delicate people, people with organic heart disease or feeble circulation, or epileptic cases dependent upon organic disease of the brain, stand bromides very poorly. Under all these circumstances the drug should be administered with the greatest care, as unpleasant, if not dangerous, effects may be produced if its use is persisted in.

Even after having decided upon what is apparently an efficient dosage in a given case, it is not wise to let the patient keep on with the drug without medical supervision. There are circumstances that indicate diminution or increase of dose. In the case of any intercurrent illness, medical or surgical, the large doses are not so important, for illness and surgical operations and accidents are of themselves most efficient anti-epileptics. The drug should not be discontinued, however, excepting in the case of an acute illness of a most depressing character. Patients, who are subject to exacerbations and ameliorations of attacks in certain seasons of the year, should have the dosage regulated accordingly. Increasing age and size of young epileptics will require a gradual increase in the dose. An extra dose should also be administered when patients have experienced any unusual excitement or fatigue.

If all the above rules are carefully followed, I am satisfied that bromism will, in a great measure, be robbed of its terrors. Unless they are followed in the minutest particular, the best results from the drug will not be obtained.

There has been much discussion as to which of the bromides is the best. So far as the potassium, sodium or ammonium salts are concerned, I do not think there is much choice. Some cases seem to get along better on one than the others. Of late, I have used largely the *bromide of strontium*, and have felt that it gives better results with decidedly less interference with the digestive functions.

Seguin has recommended, in case of the failure of the bromide to control the seizures, that a portion of that drug be replaced by a certain amount of chloral. His rule is to substitute five grains of chloral for every ten grains of bromide displaced.

Bromides are not as efficient in controlling the mild epileptic attacks as they are in the case of the convulsive ones.

As to the bad effects of bromide upon the mind, I think, as I have already hinted, that they are largely overdrawn. The great trouble is that epilepsies are not properly differentiated. Certain types are invariably associated with the highest degree of mental degradation, while cases exist in which the most brilliant intellect is to be found. It has been said that Julius Cæsar and Napoleon Bonaparte were both epileptics. These cases, however, are exceptional. The natural tendency of epilepsy

is to produce mental deterioration proportionate to the severity of the disorder. This fact was well recognized many years before the bromides were introduced as medicine. One should not be too ready, therefore, in ascribing the dementia in a given case to the effects of the treatment, rather than to the course of the disease.

No disease has more bones of contention as to its treatment than has epilepsy. Not only the bromides but the surgical measures proposed for its relief have been discussed, often indeed with a spirit that is far more acrimonious than scientific. There can be no question that some cases have been cured by the removal of reflex irritation, but these are exceedingly few and far between.

The brilliant successes of abdominal surgeons have led to indiscriminate oöphorectomizing of epileptic patients, a practice that, so far as I am aware, has not been fraught with one cure, excepting in some few cases where there was severe ovarian disease, or in which the convulsions were *limited* to the menstrual period. I consider it a practice that should be condemned in unqualified terms to remove *healthy* ovaries for the cure of epilepsy and other neuroses. Practical experience of the best and most experienced gynæcologists and neurologists bears out this view. It is true that cases of cure have been reported in medical journals, but the reports have usually been made altogether too soon after the operation. No case of epilepsy can be considered as having been cured by an operation until two years have elapsed without a fit of any kind.

The general impression as to the prognosis of traumatic epilepsy treated by operation is decidedly incorrect. Books teach and physicians believe that a trephining in a case of traumatic epilepsy is tantamount to cure. No greater error was ever promulgated. A small percentage of traumatic epilepsies are permanently cured by trephining. Nearly all experience a temporary relief from the operation. This relief, however, is merely that which follows any surgical procedure done on an epileptic. I have taken the pains to read over the reports of a large number of traumatic cases operated, and I must say that the same degree of carelessness manifested in other reports would bring upon the author a well-deserved rebuke. Thus I find an article headed, "Epilepsy of Ten Years' standing; Trephining; Cure." Reading the article, I find that the convulsions had evidently occurred as the result of an old traumatism, probably a fracture; that the case had been trephined at the seat of fracture, and that up to the time of leaving the hospital there had been no recurrence of the seizures. It is surprising, moreover, to find that the men making these incomplete reports are men of intelligence and learning. Occasionally, however, we find reports of cases really cured. While I have not analyzed the reports sufficiently to give statistics, I feel safe in estimating the proportion of cures of traumatic epilepsy at not over 20 per cent., and I would not be surprised if they did not reach 10 per cent.

Results might be better if the cases were better selected; in fact, I am sure of it. In a general way it is a safe rule to trephine all cases at the seat of injury in which the relation of cause and effect between the injury and the epilepsy is well established. We have not much reason to expect a favorable result unless the convulsions present characteristics that indicate a local seat of disturbance. In every case we cannot be too careful as to how much we promise.

Sachs endeavors to account for the unfavorable results from trephining for epilepsy on the theory that a secondary sclerotic condition is set up by the injury. There is much to favor this view. He thus explains, I am satisfied, those cases of traumatic epilepsy which are apparently dependent upon a general cerebral disturbance.

Sachs's remarks at once raise the question of prevention of epilepsy after head injuries. If we cannot cure traumatic epilepsy, can we prevent it? I do not think we know enough to answer this question. It has been said, by whom I do not now just recall, that 50 per cent. of head injuries develop epilepsy in later years. I have before quoted this remark approvingly. More mature consideration leads me to doubt it; but the percentage is undoubtedly large. Let the figures be what they may, our course is the same. Every case of fresh cranial injury should be treated according to principles that will give the parts the best chances for perfect healing. I believe it to be a good rule to trephine all fractures where there is the slightest evidence of brain injury. There may be no depression of bone, but there may be a sub- or extra-dural hæmorrhage, attention to which is an important matter. On the other hand, one must not promise that trephining will certainly prevent epilepsy, for cases have been reported in which this simple operation itself has been the traumatism that started up the disease.

Early operation should be performed as an exploratory procedure primarily. Secondarily, it acts beneficially by relief of tension.

Aside from traumatic cases, trephining has been recommended in certain types of epilepsy presenting a local origin. It has also been recommended as an empirical measure in idiopathic cases. All cures reported in the latter class are not satisfactory, the reports having been made too soon after operation. Sometimes most remarkable primary improvement occurs. This was well illustrated in a case recently under the care of Dr. Van Lennep and myself. The child had been having thirty to forty convulsions daily. Large doses of bromide given in order to suppress as far as possible the general character of the fits leaving the special character still predominant, only served to reduce the number to fifteen or twenty daily. A trephining over the middle of the fissure of Rolando, on the side opposite to that on which the convulsions were most marked, was performed. In the two months after the operation the child had but two attacks, and they were within the



first week or two afterwards. I do not know what the final result has been.

Jacksonian epilepsy is the form of the disease in which surgery has been offered as the sovereign remedy. Experience has not borne out the promise of good things made. Improvement has followed in most cases; but relapses have been common. The paralyses following the excision of a cortical centre have, in most instances, been temporary. The late histories of those cases in which I have been interested have not been what was hoped for.

Ligation of the vertebral arteries and other operations have been recommended, but have been abandoned.

Thomson, of New York, has recommended the employment of a red-pepper pack as a therapeutic measure. It is supposed to produce general peripheral irritation and exert the same influence as major surgical operation.

Actual cautery applied to the head has not been without its adherents. Falling against a stove while in a fit has effected temporary cures.

There is one thing to say concerning operative treatment of epilepsy. So much is expected from the operation itself that nothing is done afterwards to maintain the improvement. *This is a serious mistake.*

Epilepsy undoubtedly sometimes recovers spontaneously. As I pen these lines a medical friend tells me of three cases which he knows were permanently cured by time, diet and well-regulated habits.

In the preceding pages I have given my ideas as to the proper treatment of epilepsy. Let me say that they are the result of experience in managing the pick of obstinate cases. But few of them have been of recent origin.

## CONVULSIONS.

**Synonym.**—Eclampsia infantum.

Convulsions are to be regarded solely as a symptom, an indication of the disturbance of the normal health standard. Very frequently they occur as the result of some morbid condition the existence of which is very clear; but just as often their origin is involved in such obscurity as to make the recognition of their cause possible only after careful study. The diagnosis of "convulsions" is only permissible therefore in cases in which the origin of the trouble remains undetermined. These must become fewer in number as our knowledge increases. The greatest difficulty is encountered in the study of the convulsive seizures of children; and it is in these little ones that convulsions are most frequently encountered, and in whom a specific treatment for the spasms themselves is most frequently required. Children are especially predisposed because of the incomplete development of their nervous systems, by reason of which the lower centres exert too much control, or the higher cerebral centres fail to exert their normal inhibitory influence.

It is under the age of two years especially that most cases occur, although convulsions may appear at any time. Occurring during the first few days of life, they are, in the majority of cases, symptomatic of injury during birth, also of congenital pathological conditions, *i. e.*, heart disease, atelectasis, etc. It has been said by several authors that more cases occur in boys than in girls. While this statement is evidently true, it finds its origin in the fact that during the time for which statistics were taken, the male births exceeded the female in the same proportion.

The inheritance of a neuropathic constitution is a powerful predisposing factor. One not infrequently meets with families in which the parents are highly neurotic, and in which all the children exhibit a convulsive tendency in response to the slightest exciting cause. It has been very properly held that a healthy child cannot have convulsions from reflex irritation alone.

Of constitutional predisposing causes, rachitis stands high. This fact obtains more and more recognition each succeeding year. Rachitic children are especially liable to laryngismus stridulus. The explanation of the convulsive tendency of rachitic children is found in the alterations in the blood supply to the brain, to the deformity of the ribs interfering with proper respiration, and therefore, with proper oxygenation of the blood, to general failure of nutrition, and to cranio-tabes. As to the causation of the latter condition, there has been considerable discussion,

some regarding it as a rachitic condition pure and simple; others claiming that it can exist only as a syphilitic phenomenon. In this condition there is thinning of the cranial bones, especially the occipital, in patches, and the slightest pressure exerted thereon, even that of the pillow, is sufficient to excite a convulsive seizure.

Among the laity dentition is regarded as the great cause of convulsions. This is a mistake. The belief arises from the fact that delayed dentition is often but one of the symptoms of rachitis. Rachitis generally asserts itself between the sixth and eighteenth months, and it is at this time that dentition should be active. The period of dentition is, moreover, one of increased functional activity, during which exciting causes take on increased activity. Indigestion is liable to occur by reason of the changes going on in the structure of the intestinal follicles to prepare them for a change of food. The folly, therefore, of resorting to gum lancing is rendered apparent.

Of all exciting causes *gastro-intestinal irritation* stands first. In the majority of cases in which convulsions arise from local irritation, improper feeding, either the ingestion of too large a quantity, or the taking of improper quality of food, is responsible for the entire difficulty. Gastro-intestinal diseases, as diarrhoea, may give rise to convulsions.

*Intestinal parasites* are a somewhat frequent cause of infantile convulsions, though not as frequently so as lay opinion would lead one to believe. Lumbrici are the most influential in producing the necessary intestinal irritation. Thread worms very rarely cause convulsions.

A very common cause of infantile convulsions is found in the onset of the *acute infectious fevers*. Sometimes it appears that the convulsion takes the place of a rigor, while at others it occurs apparently from the intense toxæmia or the constitutional effects of the high temperature. In intermittent fever in infants the convulsions frequently displace a chill. Such attacks are very severe, and are a source of great danger to life. Convulsions sometimes usher in acute poliomyelitis anterior.

Convulsions may be sympathetic of various *toxæmia*, either as already stated, the result of infectious diseases, or from such conditions as uræmia, the absorption of ptomaines, or the ingestions of a variety of poisons.

*Whooping cough* is sometimes a cause, it acting through exciting venous congestion of the brain, its occasional high fever, or its associated nervous elements.

*Malformations of the heart* constitute a cause in the early days of infantile life.

*Peripheral irritations* in great variety are causative factors. Burns and scalds are frequently operative etiologically. Otitis media sometimes excites convulsions, not only because of the reflex influence of the pain, but also because of the proximity of the inflamed parts of the brain.

*Violent emotions*, as fright, anger, etc., causes spasms in children.



It is even believed that the same causes in the mother may excite the trouble in the nursing infant through changes thereby occasioned in the milk.

Convulsions are often symptomatic of serious disease of the nervous system. Thus many cases arise in the early days of meningitis, or usher in attacks of paralysis, both cerebral and spinal in origin. Many cases of epilepsy are in their incipency looked upon as simple reflex spasms.

**Symptomatology.**—An attempt to describe the symptomatology of a symptom is certainly an inconsistency. At the same time there are phenomena attendant upon convulsive seizures worthy of note under this heading. The attack may come on suddenly without the slightest warning, during either the sleeping or waking state. Just as frequently the child gives evidence for a greater or less length of time of nervous disturbance, as starting and twitching during sleep, unusual irritability, or brightness, etc. The convulsion itself consists of tonic and clonic spasms, and is not distinguishable by its movements from the ordinary epileptic seizure. The whole or part of the body may be involved. During the continuance of the movements the face is livid or pale. On the subsidence of the attack the patient passes into a sleep or stupor which may last from a few minutes to several hours.

**Duration, Course, etc.**—An attack of convulsive attack may be over in a very few seconds, or it may last several hours. It may consist of one or many spasms. When the result of peripheral irritation they are generally single and of short duration; *i. e.*, they last from a few minutes to half an hour.

**Diagnosis.**—The great problem in the study of any case of convulsion is the determination of the cause of the attack, and the differentiation of functional from organic cases. The most important question in this connection is the differentiation of simple idiopathic convulsions from epilepsy. It is a noteworthy fact that many cases of the latter disease make their appearance during infancy and early childhood, and are then regarded by the physician as of slight moment. It should be ever kept in mind that any case of convulsion, no matter how certainly reflex in origin, may possibly be epileptic. Even more than this, reflex convulsions occurring repeatedly may engender the convulsive habit and thus give rise to epilepsy, attacks of which occur notwithstanding, the removal of every possible source of irritation. Functional spasms from reflex irritation are very rare after the age of three years. Convulsions occurring after that age must therefore suggest the possibility of epilepsy very strongly. In favor of epilepsy at any age is the recurrence of convulsions coming suddenly when the child is in the best of apparent good health, without any warning other than an aura, lasting but a short time (five to fifteen minutes), and leaving the patient in as good a condition as before the seizure.

Convulsions arising from fright, overloading of the stomach, rachitis, etc., are to be recognized by the history of the case. The ushering in of an attack with high fever suggests the onset of some acute infectious disease.

Cases dependent upon organic disease of the brain display their true nature by the local distribution of the convulsive movements, and the great length of the attack, which often leaves the patient paralyzed.

**Prognosis.**—Reflex convulsions are rarely fatal. They are somewhat more dangerous than epileptic attacks. Convulsions occurring in the course of severe diarrhœa and other exhausting diseases must be regarded as dangerous complications. Convulsions symptomatic of organic brain disease are also of serious omen. Frequent convulsions may cause death by exhaustion.

**Treatment.**—Much can be done in the way of preventing the convulsive tendency in those predisposed to attacks. Rachitic and other illy-nourished patients must be well-fed and carefully nursed. At the same time the greatest care must be exercised lest the stomach be overloaded. Regular feeding with easily digestible food should be the rule. Milk and milk foods constitute the best article of diet for these little patients. Heavy table food must not be permitted. Fresh air and plenty of it is necessary for the rachitically inclined. When the attack is evidently the result of gastric or gastro-intestinal irritation, the stomach and bowels must be emptied at once. The child may be made to vomit very promptly by irritation of the fauces with the fingers. The bowels should be washed out with a large enema. The little one should be kept absolutely quiet in a darkened room. If the convulsions continues more than half an hour, then recourse may be had to chloroform anæsthesia. This procedure is perfectly safe if applied intelligently, as but little of the drug is required to produce an effect in children.

When the temperature is high the child may be treated to a cold douche, and an ice-bag may be applied to the head. The warm bath has obtained a world-wide reputation as a panacea in convulsions, and is in general use among the laity. As long as the water is used warm, it cannot be productive of harm. It may be permitted therefore, but it is not likely to do any good.

*Ignatia* enjoys a greater reputation in the treatment of convulsions than does any other remedy. It is especially indicated in those cases occurring after fright or other violent emotions. The movements are largely of the tonic order, such as observed in strychnine poisoning. The child may have exhibited a neurotic tendency for several days preceding the attack; *e. g.*, there may have been whimpering during sleep. The face is usually pale during the convulsions, though it may at times be flushed.

*Belladonna* is indicated in convulsions arising from anger or violent

emotions, and from reflex irritation. The symptoms suggesting its use are the bright red flushed face, the hot head, the wild staring eyes with dilated pupils, throbbing carotids, pungent heat of the surface of the body and spasm of the glottis.

*Glonoïn* is to be employed where there is evidently violent congestion to the head, and when arterial tension is high. Like belladonna and opium it is indicated for the bad effects of violent emotions.

*Cuprum* has convulsions associated with marked blueness of the face and mouth. The hands and fingers are firmly closed.

*Opium* is useful for cases occurring after fright; but there is a turgidity of the face, and stupor following the movements is profound.

*Aconite* and *veratrum viride* are useful in cases in which the circulatory disturbances of those remedies are present.



## ASTASIA-ABASIA.

Astasia-abasia must be regarded as a term designating a group of symptoms, and not a special disease. As to the symptoms making up this group, authorities seem to differ. Gowers allies the condition with vertigo, saying: "The sufferer from it, when walking, will suddenly fall to the ground (astasia), often forward; it seems to him that his legs give away. When sitting, he will suddenly bend forward, his head dropping on the chest, and his body seems to lose its power of support (abasia). In either case, in a minute or two, normal strength returns. There is never loss of consciousness."

Charcot, Blocq and their followers use the term to designate a condition in which the patient can neither walk nor stand, notwithstanding motor power, co-ordination, and sensation are unimpaired. In some cases, the impression given the examiner is that the difficulty is paralytic, in others, that it is ataxic, and in still others, that it is spastic.

The majority of cases are associated with hysteria, while many others give a history of other functional neuroses, as epilepsy, chorea, and psychoses.

While the patient is lying in bed, she seems capable of performing any movement with strength and precision. It seems more than probable that it is of a purely hysterical nature.

**Prognosis.**—This is favorable, most cases making a good recovery.

**Treatment.**—This must be conducted on the same principles as outlined in the chapter on hysteria.

## AKINESIA ALGERIA.

**Synonym.**—Pain palsy.

**Definition.**—Akinesia algeria is a condition first described by Möbius, characterized by inability to move because of pains in the part, the existence of the pains being unexplainable on any other hypothesis than that of their psychical origin.

**Etiology.**—The disease or condition, whichever it may be called, is of very rare occurrence. It seems to affect only persons of highly neurotic constitutions, especially those the subjects of paranoia. The patients thus far have all been adults.

**Symptomatology.**—Akinesia algeria is of gradual onset. Usually the patient gives a history of various psychoses extending over a term of years. Pains come on and are complained of as being intense. At the same time, the most careful examination fails to account for their presence. They are believed to be hallucinations. They are greatly aggravated by the slightest motion, which, indeed, causes the most intense suffering. The patient therefore lies in bed motionless and helpless. There is no muscular atrophy. Sensory symptoms are absent, and the electrical reactions are normal. No alterations in the reflexes have been found.

**Pathology.**—The disease is believed to present no pathological changes discoverable by means at present at our disposal. The pains being the product of hallucinations, akinesia algeria must be regarded as a form of insanity. It hardly seems to be a variety of hysteria.

**Prognosis.**—These cases generally last for many years. They are always obstinate to treatment, and are often incurable.

**Treatment.**—The current belief is that these cases are best managed by the judicious application of rest, feeding and seclusion.

# TROPHIC AND VASO-MOTOR AFFECTIONS.

## ACROMEGALY.

**Synonyms.**—Akromegaly; acromegalie; Marie's disease.

**Definition.**—A disease characterized by progressive enlargement of the extremities, the hands, feet and certain bones of the face being especially liable to the morbid changes.

The recognition of acromegaly as a separate clinical entity is of very recent date, it having been first described by Marie in 1886. Prior to that date there had been a number of cases which we must now regard as examples of this disease, reported under various designations, principally, however, as instances of gigantism, exophthalmic goitre, myxœdema, etc. Medical literature now includes reports of over one hundred cases of acromegaly, some few of which are doubtless cases of mistaken diagnosis. The clinical history of the disease is such as to make its recognition a mere accident. Dercum's cases were discovered by the merest chance. My two cases may also be regarded as discovered by accident. One came to my clinic for treatment of his headaches, after having been the round of hospitals. The second one entered the Hahnemann Hospital because of suspected renal calculus. In both the history of progressive enlargement of the hands and feet and the characteristic changes in the features were clear.

The causes leading up to the production of acromegaly must be regarded for the present as unknown. Osler, following the reports of Souza-Leite, gives sexual predisposition of the disease to the female. Studies of all cases to date show that the cases are about equally divided between the two sexes. The majority of cases begin in early adult life, that is, between the ages of twenty and forty.

**The Pathology and Morbid Anatomy** of this interesting affection likewise seem to be unfathomable. In the present state of our knowledge we cannot describe any special changes other than those occurring in the bones. An enlargement of the thymus gland has been found by Klebs and Fritsche. The thyroid gland has been found hypertrophied, normal, and atrophied. Klebs and Gauthier look upon the disease as an angiomatosis. Von Recklinghausen assigns acromegaly a neurotic origin. In a number of autopsies the pituitary body has been found enlarged. This has led to the theory that the disease originates in this



lesion. Such a theory is without good foundation, for cases of acromegaly have existed in which the pituitary body was normal, and instances of tumor in that locality have been reported in which trophic changes in the extremities were absent.

**Symptomatology.**—The characteristic condition leading to a diagnosis of acromegaly is a progressive enlargement of the hands and feet and of the projecting portions of the face. All the tissues in the affected parts, bones, cartilages, connective tissue and skin participate in the hypertrophy. As already stated, the disease is usually discovered by accident, the patient consulting a physician for some annoying symptom, as headache, amenorrhœa, etc. Investigation then reveals the fact that for several years the patient has been wearing progressively larger and larger gloves and shoes.

Sexual symptoms are among the earliest to appear. In women there is amenorrhœa, and in men gradual loss of sexual power.

The hypertrophy of the extremities is observed in the hands and feet. Sometimes the wrists participate in the change, but the long bones of the arms and legs remain normal. Sometimes there is an enlargement of the patella. The fingers enlarge equally through their entire length, but in thickness only, giving them an appearance described as "sausage-shaped." The toes enlarge in like fashion, the great toe often, however, increasing in size out of proportion to the others. The only impairment in the function of the hands and fingers is found in the unwieldiness naturally following such an increase in size.

The changes in the head are confined to the cartilages of the ear, nose and eyelids, and the superior maxillary, malar and inferior maxillary bones. The ear becomes greatly increased in size; the nose enlarges, and the eyelids grow thick. The hypertrophy of the superior maxillary and other bones causes a lengthening of the face out of all proportion to the size of the cranium. The inferior maxillary bone increases in size in all directions. Thus it projects beyond the superior maxillary, making quite a space between the upper and lower teeth in attempted articulation of these structures. The alveolar border undergoes thickening. The teeth, not increasing in size with the jaw, become separated. The supraorbital ridge very often grows prominent, thus giving the forehead a retreating appearance. All these changes in the face give to it an oval shape, the greater curve of the oval being below.

Changes in the sternum, the bones of the pelvis, the bones of the thorax, and the vertebral column, are likewise present. The manubrium sterni is thickened. This leads to dulness on percussion in this locality, a phenomenon which Erb attributed to enlargement of the thymus gland. The xyphoid cartilage stands out prominently. The enlargement of the pelvic bones is especially noticeable about the crests of the ilii and the pubic arch. The changes in the vertebral column lead to

a kyphosis in the cervico-dorsal region, which depends upon thickening and ossification of the intervertebral cartilages.

Subjective symptoms are by no means wanting. The most constant of these is the headache, which is but exceptionally absent, and is nearly always terrific in character. Various paræsthesiæ are observed in some cases. These include sense of numbness, formication, etc. The mind may or may not be impaired. When affected, it is usually in the direction of loss of memory.

The tendon reflexes are usually normal, but they may be absent, as in both of my cases.

The skin is thick and harsh; the hair is thick and coarse. The tongue is thick, broad and large, leading to impairment in speech.

Attention has been directed of late to visual disturbances in acromegaly. These may consist of mere blurring of sight, or they may amount to such serious affections as restriction of the visual field to the extent of hemianopsia, or atrophy of the optic nerve.

In some cases inordinate somnolency has been a prominent symptom.

**Diagnosis.**—The diseases with which acromegaly may be confounded are simple gigantism, myxœdema, osteitis deformans, osteoarthropathie pneumique, and local hypertrophies. *Gigantism* presents a uniformly large growth throughout the body, no one organ or part being disproportionately larger than any other.

The resemblances between *myxœdema* and acromegaly are superficial. Myxœdema occurs in the majority of cases in women; acromegaly affects the sexes equally. Myxœdema is a disease of middle life; acromegaly, of young adults; the bones are never involved in myxœdema; the face in myxœdema was described by Sir Wm. Gull as round, like the full moon; the face in acromegaly is oval. In myxœdema the ends of the fingers are clubbed; in acromegaly the fingers maintain the same enlarged size throughout. In myxœdema the swelling is due to subcutaneous infiltration.

*Osteitis deformans* also bears but a superficial resemblance to acromegaly. It is generally a disease of middle life; the long bones of the extremities are principally involved, and they are not infrequently curved, thus adding to the deformity. The bones of the cranium are affected. The parts affected are by no means symmetrical.

*Osteoarthropathie pneumique* is a condition described by Marie in 1890. It consists of an enlargement of the extremities accompanying disease of the lungs. The changes are observed in the joints and ends of the fingers. The large joints are also affected, and motion is impaired. The nails are curved and clawed. The soft parts are not involved in the hypertrophic process.

*Local hypertrophies* are readily distinguished by their very nature,

**Prognosis.**—It is believed at present that the prognosis of acromegaly is unfavorable. After a duration of from ten to twenty years the patient falls into a cachectic condition, the muscles atrophy, and he dies of sheer exhaustion. In some cases the tumor of the pituitary body causes coma and death.

**Treatment.**—This of course is very unsatisfactory, and must be directed to making the patient as comfortable as possible. Fowler's solution gave great relief to one of my cases and has been reported as benefiting cases in the care of other observers. Thyroid feeding has been advocated, but thus far has seemed to accomplish little or nothing.

*Sulphur* and *silicea* prescribed symptomatically greatly helped the case reported by O'Connor.



## SCLERODERMA.

Scleroderma is a rare affection, characterized by marked induration of the skin occurring in patches or over a diffused area. Neither its pathology nor etiology is at all understood. It is a matter of observation that most cases occur in adults and in women. In one case reported by Rabl a wound appeared to be the exciting cause. Kaposi, from a study of 175 cases seen by him, reached the conclusion that nervous influences of some kind were almost invariably at work. Exposure to cold and the rheumatic constitutions have been assigned as causes, some authors regarding these influences as of the greatest etiological importance. Pathologically, a hyperplasia of the fibrous element of the papillary layer and corium of the skin has been found. At the same time there is a decrease of the subcutaneous fat and increase of pigment deposit. Hoffa finds, in addition, a perineuritis and endarteritis fibrosa.

**Symptomatology.**—Scleroderma has been divided into two varieties, the circumscribed and the diffuse, *scleroderma localis* and *scleroderma diffusa*. The former is ushered in by neuralgic pains and nervous symptoms as a rule. The local changes appear insidiously and consist of patches of induration of the skin, occurring in patches or in striæ, extending the length of the affected limb. This variety of scleroderma is considered by some authors as closely identified with morphoæ and the keloid of Addison.

*Scleroderma diffusa* is characterized especially by the symmetry of the lesions, which, beginning as a rule on the back of the neck, extend laterally. Still they may start in the extremities, in which case symmetry is still their prominent feature. The induration is almost a stony hardness; the affected skin is united closely to the subjacent structures. The underlying muscles undergo atrophy. Naturally the limbs become stiff and their movements impaired. Sensation is not affected. The patches exceptionally remain normal in color; usually, however, there is an excessive deposit of pigment, this pigmentation often preceding the induration. Sometimes the lesions, by mechanically obstructing the circulation, produce œdema of the hands and feet. The hair and nails are not as a rule affected.

Scleroderma runs a very chronic course, covering a period in several instances of twenty years. It does not directly lead to a fatal issue. The patient seems to be predisposed to pulmonary affections, which result fatally.

The most important element in the treatment of these cases is pro-

tection from changes of heat and cold. The patient should be properly clad. Blocq has reported a cure effected by electrolysis. Needles were inserted obliquely into the indurated parts. He claims that the beneficial effects of the current were observable in parts not directly subject to electrolytic action.

The remedies adapted to these cases are *bryonia*, *guaiacum*, *graphites*, *hydrocotyle*, *lachesis*, *phosphorus*, *silicea*, and *stillingia*.

### SCLEREMA NEONATORUM.

Sclerema neonatorum is a condition in the new-born in which the skin becomes stiff and indurated and assumes a white or waxy hue. It may be either congenital or acquired. In the former case, the child is usually still-born. In the latter, the first changes in the skin are observed three or four days after birth, and generally on the lower extremities. From the initial point of attack the induration extends to all parts of the body, even becoming so extensive as to make motion at all the joints impossible. The pulse is slow, and prostration becomes profound. Death sets in on about the sixth or seventh day of the disease.

The treatment is the same as that recommended for scleroderma.

### AINHUM.

Ainhum is, probably, a variety of scleroderma, in which a band of indurated tissue forms around one of the toes or extremities, and by its interference with the nutrition of the parts beyond, causes death and separation of the same. It was first described by da Silva Lima, in 1867, and was for a long time supposed to be a disease limited to Brazil, and the negro race. Cases have exceptionally been observed in the white race, and in other countries. The course of the disease is usually a progressively slow one. Sometimes, however, spontaneous arrest of the morbid process takes place. The treatment consists in division or excision of the constricting band.

## HERPES ZOSTER.

**Synonyms.**—Zona; shingles. The popular name for this disease is generally regarded as a corruption of the Latin "*cingulum*," a girdle. Popular tradition for ages has held that when the eruption of shingles completely circles the body, death must ensue; a tradition that is based on fallacy, for so far as known this disease has not proven fatal.

**Definition.**—A disease characterized by the appearance of vesicles in groups along the course of a definite nerve trunk, and associated with changes in that nerve.

**Etiology.**—The causes of herpes zoster are as varied as they are interesting. *Traumatism* of a nerve not infrequently gives rise to a condition bearing a close resemblance to this disease, the eruption usually manifesting itself within a few hours after the reception of the traumatism, and following the course of the nerve or plexus of nerves injured.

*Gouty predisposition* is assigned as a very important cause by some; indeed it has been claimed that in the absence of this, traumatism of nerve trunks is incapable of producing zoster.

Some cases have been known to occur after *exposure to cold*, or after violent emotions, as anger. In the latter case the trouble has followed the cause almost immediately.

Hutchinson and Wm. Oliver Moore have observed cases in which all the evidence favored the view that the disease arose from Fowler's solution, which the patient happened to be taking at that time.

Erb and Landouzy believe in the *infectious origin* of herpes zoster, and attribute its existence to a specific germ. There is nothing in support of this theory beyond the fact that when zoster occurs in hospitals devoted to the treatment of diseases of the skin, numerous other cases are observed at about the same time.

*Syphilis* seems to be a cause in some few instances. The eruption is then bilateral and symmetrical.

Neither sex is any more predisposed to the disease than is the other. It affects all ages.

**Symptomatology.**—The disease may or may not be preceded by prodromic symptoms. In the latter case there appear such indefinite symptoms as headache, nausea and general fatigue. Fever is almost invariably absent. There is next felt a burning sensation or neuralgic pain in the part on which the eruption is about to appear, usually over the course of one of the intercostal nerves. In the course of from twenty-four to forty-eight hours the eruption manifests itself first as small red



points arranged in oval groups, and these rapidly go on to vesication. The contents of the vesicles are at first clear, subsequently becoming translucent, and finally opaque from the admixture of pus. In from ten days to two weeks the eruption begins to dry up, scabs form and fall off, leaving red spots or cicatrices. Very often permanent scars remain.

The eruption presents the same general characteristics, whatever be its situation. Occurring in the course of the trigeminal nerves, it is very frequently mistaken for erysipelas, from which it differs in the distribution of the lesions, and by the appearance of perfectly healthy skin between the vesicles. Sometimes the eye is affected, in which case ulceration of the cornea and iritis exist as complications. It was pointed out by Hutchinson that the eye is not affected excepting when the nasal nerve is involved.

The most annoying symptom of zoster is the neuralgia which either precedes or follows the eruption. In young subjects this is very frequently absent. In adults it is commonly present, and in those of advanced years it is not only very constant, but very severe and obstinate to treatment.

**Prognosis.**—The prognosis of herpes zoster is always favorable. In old and cachectic subjects the neuralgia may persist for a long period; indeed, it may seem almost incurable.

**Treatment.**—The only local medication of value is that which protects the vesicles from the irritation of the clothing, which may best be done by the application of vaseline. As internal remedies *rhys*, *mezereum*, *ranunculus bulbosus* and *arsenicum* are the most important. *Rhus* is especially suited to the cases arising from traumatism or exposure, and in which the predominant sensory symptoms consist of itching, burning and tingling. *Mezereum* is adapted to zoster affecting the trunk, associated with severe neuralgic pains. These are, as a rule, aggravated at night and when the patient is in bed, and are followed by a numbness in the affected parts. It is indicated even in cases in which the neuralgia is the sole symptom, the eruption having disappeared. *Ranunculus* is regarded as a remedy more suited to cases in which the supraorbital nerve is effected; the pains are of a sharp, stitching character, and the fluid contained within the vesicles is of an excoriating nature. *Arsenicum* is a valuable remedy for the subsequent neuralgia, for which it may be given even in officinal doses of Fowler's solution. While these remedies will be found adapted to nearly all cases, others may occasionally be required, in which case *graphites*, *iris*, *croton tiglium*, *cantharis*, *euphorbia* and *sulphur* should be studied.

## ANGEIONEUROTIC ŒDEMA.

**Synonyms.**—Acute circumscribed œdema; giant urticaria; urticaria tuberosa; acute non-inflammatory œdema.

**Definition.**—An acutely appearing œdema, either circumscribed or general, occurring suddenly, as the result of vaso-motor disturbances.

Angeioneurotic œdema is a disease but recently described in medical literature, although evidently known in a general way for years. It is usually spoken of as a rare affection, and yet there are but few physicians of any experience who have not observed numerous examples of it. Now that the study of the affection has been begun, increasing knowledge will shortly show that under this heading are at present included quite a variety of œdemas, all of them non-inflammatory in origin, and independent of renal, cardiac, or hepatic diseases.

**Etiology.**—The causes of angeioneurotic œdema seem in a general way to be identical with those of urticaria, with which disease it appears to bear an intimate relation. It occurs especially in neurotic subjects, by preference in young adults, though no age is exempt. Sometimes it is hereditary, Osler reporting a most remarkable instance in illustration of this statement. It likewise occurs without regard to the condition of general health, the sickly and the strong being alike liable to it. The exciting causes are mostly those of urticaria. Exposure to cold, partaking of certain articles of food, blows, anxiety, and traumatism, have brought on attacks.

**Symptomatology.**—A detailed description of the symptoms of angeioneurotic œdema is unsatisfactory, because cases vary greatly in their clinical features. All begin suddenly, but present the widest variations in their subsequent careers. One type of the disease was well illustrated in a case seen by me in 1889. The patient was a married woman, twenty-six years of age, who, with the exception of some uterine disease, was apparently in good health up to the time she was seized with the ailment. It made its appearance rather suddenly one morning with severe headache, swelling of the entire body and high fever. Later she began to feel as if bruised all over. She had a temperature of 104.5° F.; the entire person was extremely sore to touch; the headache from which she was suffering was so severe as to deprive her of all sleep; the urine was scanty, and loaded with urates. Her most remarkable symptom was the all but universal œdema; the eyelids were swollen shut; the arms and legs were swollen; the conjunctivæ were so highly chemosed as to almost protrude from between the lids. Apis 1x was prescribed. The

temperature was but 99° on the following morning, and in a few days the patient was entirely well.

As already stated, the œdemas from vaso-motor disturbance exist in great variety. Probably the most frequently met with is the acute circumscribed œdema of Quinke. This author in describing the affection says that it bears a striking resemblance to giant urticaria. I have already suggested that it is not seldom dependent upon the same causes as urticaria for its being. This statement I have made because of having seen one case of this affection in a young lady of twenty-three, who had frequently suffered from urticaria. One morning she awoke with a stiff swollen feeling of the face, due to œdema of that part. The urine was perfectly normal. In the afternoon the œdema disappeared and urticaria appeared in the extremities. There was no itching of the face, in fact no abnormal sensations except the tense swollen feeling referred to. In describing this affection, Quinke says that the malady manifests itself by the appearance of œdematous swelling of the skin and subcutaneous tissues in circumscribed spots, from two to ten centimetres in diameter. While they generally appear upon the extremities in the vicinity of joints, they may also invade the body and face. When involving the latter locality, they are especially prone to attack the eyelids and upper lip. The swollen portions of the skin are not, as a rule, reddened, but are pale and translucent in appearance and not sharply defined. The only subjective sensation present is one of tension in the swollen part, itching being but rarely felt. Not only the skin, but the mucous membranes may be involved. Thus the mucous membrane of the larynx may become so œdematous as to give rise to sudden dyspnœa, and even death. In one case certain symptoms of the stomach and bowels suggested that these organs were involved in the pathological process. The œdema usually appears simultaneously on various parts of the body, and after an existence of from several hours to a day, disappears almost as quickly as it came. Successive crops of the œdema may appear, and thus prolong the duration to several days or even weeks. Throughout the course of the affection the general health is undisturbed. Occasionally an indefinite prodromal indisposition may be experienced, or during the attack itself, heaviness of the head, thirst and diminished excretion of urine. A rise in the temperature of the body has never been observed. Most of the patients exhibit a nervous, irritable temperament.

A case of acute circumscribed œdema having a very unusual cause is reported by Matas. The patient was an Italian woman who had had no other ailments in her life than childbirth and malaria. She was affected with a peculiar disfigurement of the face, which came on each day between the hours of 8 A.M. and 1 P.M. It consisted of an œdematous condition of the upper lip, which caused that part to rise to the level of the nostrils, obstructing the nares and projecting two or three



inches beyond the surface of the teeth. The swelling came on gradually, and increased in intensity for several hours, reaching the point of maximum intensity at 1 P.M., after which it gradually subsided. The swelling was perfectly painless. A careful examination showed the heart and kidneys to be quite normal. There was no anæmia or dyscrasia of any kind. Neither the cheeks nor the eyelids participated in the swelling. Owing to the periodicity of its appearance, malaria was assumed to be its cause. Quinine was prescribed. In two days' time the whole trouble had disappeared, and did not return.

Some of the published descriptions suggest that acute circumscribed œdema may involve very peculiar portions of the body. Thus Riehl reports a case in which the œdema involved the tissues of the orbit back of the eyeball, producing marked exophthalmos. There was also in this case, swelling of the eyelids and chemosis. Meynert claims that in some cases the œdema may occur in the brain and lead to sudden death. I have always attributed the severe headache in the case described in an earlier portion of this article to increased intracranial pressure from cerebral œdema.

In some cases of acute circumscribed œdema the condition may occur only during the menstrual period. Thus Bache McE. Emmett relates the case of an unmarried woman, aged 32 years, who at each menstrual period had a localized œdematous condition confined to the left eyelids with ptosis.

Bassi reports a case of unilateral œdema recurring in an hysterical young woman at each menstrual period. There was no disturbance of the general health, although the patient was described as delicate. The œdema was limited to the right half of the body. There was no difference in the temperature, color or sensibility of the two sides. There were signs of congestion of the lung on the affected side. The right ovary was painful. Both corneæ and the fauces were anæsthetic.

Dr. Tom Robinson, in reporting a case of fugitive œdema of the eyelids in a woman passing through the climaxis, remarks that he has often seen localized œdemas in women during the menopause. These swellings appear on the hands and arms, are preceded by pain, and disappear in a few hours, or after exercise. He also directs attention to localized œdemas of the face and eyes following attacks of hemicrania.

Illingworth regards these œdemas about the lower eyelids as due to passive congestion of the infraorbital region, causing a mechanical block to the passage of the venous blood, and the consequent local serous effusion. His experience led him to assert that the œdema was associated with pain in the temporal region of the affected side.

Rheumatic œdema is a form of œdema about which but little seems to be known. It has been studied by Potain, Ferrand, Farnet, Davaine and others. It has also been described as rheumatismal pseudo-erysip-

elas. It may occur in a person the subject of acute rheumatism or the rheumatic diathesis. As an example of this affection may be quoted the case reported by Proust: The patient had general acute rheumatism, complicated by patches of erythema and purpura hæmorrhagica rheumatica. The arms were the seat of large blotches of acute rheumatic œdema. The patient died suddenly from acute intestinal obstruction. The autopsy showed that a fibrinous exudation into the meshes of the cellular tissue was the pathological change to which the œdema was due. In some cases the œdematous spots are very hard, so hard in fact as to give rise to the impression that they are tumors. The extremely variable conditions under which rheumatic œdema shows itself may make its diagnosis a difficult matter at times.

It is said that local œdematous conditions may occasionally occur and complicate the course of certain organic nervous diseases, such as tabes dorsalis and myelitis. While I have treated a large number of cases of these affections, I have not yet met with this complication. Mathieu and Voigt, who have made a special study of this subject, have come to the conclusion that these œdemas are no evidence of diffused kidney disease, but simply trophic disturbance due to the alterations in the nutrition of the skin and the subcutaneous tissues. They have noted that these œdemas frequently select for their appearance places which had previously been the seat of lightning pains.

**Prognosis.**—Acute circumscribed œdema is followed occasionally by disastrous results by reason of the infiltration occurring in the lumen of an important canal and closing the same entirely. Thus in the larynx it has produced sudden stoppage of respiration and death. Other than from this and similar accidents, it is not attended with any danger. The current belief in the old school is that angeioneurotic œdema offers an unfavorable prognosis as to permanent cure. This of course refers to the typical cases, as described by Quinke. My own experience contradicts this, for patients may be greatly benefited by treatment.

**Treatment.**—Under no circumstances must this be a specific one directed to the disease itself, but rather to the general condition of the patient. As a rule one finds the patient of neurotic habit. In the majority of such cases rest and careful attention to proper feeding will accomplish wonders. Among remedies I have used *apis*, *ferrum* and *strychnia* most frequently. Details of particulars in carrying out the above plan of treatment will be found in the articles on hysteria and neurasthenia.

## RAYNAUD'S DISEASE.

**Synonyms.**—Local asphyxia; symmetrical gangrene; *digiti mortui*; “dead fingers.”

**Definition.**—A disease probably dependent upon vaso-motor disturbances in affected parts, and manifested clinically by local anæmia, local congestions, and in extreme cases, local gangrene. These phenomena occurring in succession, constitute the fully developed typical form of the disease. Abortive forms frequently occur in which the first or the first and second named constitute the entire trouble.

The existence of the cases now classified under the name of Raynaud's disease has been recognized for very many years, but they were regarded as clinical curiosities. In 1862 Raynaud described the disease systematically, reporting some twenty-eight cases observed in the practice of himself and others. He regarded the trouble as due to spasm of the bloodvessels in the affected parts, and proposed that it be designated “*asphyxie locale*.”

**Pathology and Morbid Anatomy.**—But little seems to be known respecting the pathology of Raynaud's disease. The weight of authority is in favor of assigning to it a vaso-motor origin. Very few opportunities for autopsies occur. Neuritis has been found in some instances. Jacoby found an obliterating endarteritis. The local anæmia is undoubtedly due to contraction of the vessels, while the asphyxia or congestion is the result of dilatation of the capillaries and small veins. The gangrene naturally ensues upon a persistence of these vascular changes for any great length of time.

**Etiology.**—The etiology of Raynaud's disease is not less obscure than its pathology. It affects both children and adults. In the former it is very liable to attack portions of the trunk, and then becomes a very dangerous affection. Women seem to be more predisposed to it than are men. It is especially liable to affect the anæmic, the neurasthenic, and the illy-nourished, and it is not infrequently preceded by one of the acute infectious fevers, as malarial or typhoid fever. Exposure and fright have seemingly acted as causes.

**Symptoms.**—The symptoms are sudden in their appearance. They consist of both local and constitutional manifestations, the former in the great majority of cases being the only ones present. The latter may be regarded as consisting of two classes, first those accompanying many other diseases, and dependent upon the general effect of the local trouble, as headache, nausea, loss of appetite, etc.; and, secondly, those



resulting from vascular disturbances in various organs, *e.g.*, the brain. Thus have been noted numerous cerebral symptoms, such as great mental depression, transient loss of consciousness, acute mania, convulsions of epileptiform type, etc.

The local symptoms may be best studied by treating of them under three stages, namely, (1) local syncope, (2) local asphyxia, and (3) local gangrene. The first stage is suddenly ushered in with numbness and various morbid sensations in the extremities, especially in the fingers and toes, and often attended with pain. Objectively the affected parts are found to be pale, bloodless, cold and shrunken. Perspiration may be either excessive or absent. The affected parts are stiff, and more or less immovable. This stage of local syncope may last from a few minutes to several days. It may constitute the entire seizure, or it may merge into the second stage, that of local asphyxia. In the latter case, the spasm of the vessels is succeeded by relaxation, and there immediately occurs an intense local engorgement. These vascular changes do not occur alike in all the affected parts. It is not unusual to note syncope in one finger, while its neighbor is in a state of asphyxia.

The stage of asphyxia may occur as the primary, and, indeed, the only one of Raynaud's disease. The affected parts assume a livid or cyanosed appearance. The tissues are swollen, and the surrounding parts become œdematous. Pain arises from the local tension, and may be quite severe. Pressure of the finger on the congested portions causes local pallor. When pressure is removed the color returns more or less rapidly according to the condition of the capillary circulation. The stage of asphyxia, if sufficiently prolonged, terminates in gangrene.

The necrotic change is first manifested, as a rule, by the formation of blebs. Sometimes, however, the affected parts simply shrivel and become black; a line of demarkation forms, and the dead tissue is cast off. In this way the entire terminal phalanx, and even a hand or a foot, may be lost. Usually, the necrosis is but superficial, involving only the skin and the immediately subjacent connective tissue. As soon as the slough is cast off, granulation takes place. The parts generally affected are the bulbs of the fingers and toes, the tips of the ears and nose. A noteworthy characteristic occurring in any situation is their symmetry. The duration of the gangrenous stage of Raynaud's disease is indefinite.

**Diagnosis.**—Raynaud's disease is to be distinguished in its fully developed form from gangrene arising from other causes, such as obliterating endarteritis, ergot poisoning, senile gangrene, and frost-bite. The condition of the pulse, the history of the case, the peculiar superficial and symmetrical distribution of the gangrene, are the points on which we must rely for differentiation.

The abortive form, *digiti mortui*, requires separation from neuritis and neurasthenia. This may be done by careful physical examination.

**Prognosis.**—Very few cases of Raynaud's disease die. The greatest danger is found when the disease attacks children, and the gangrene invades the trunk. The prospects of permanent recovery are very poor. Once a patient is affected by this disease, recurrence is very probable, indeed almost certain.

**Treatment.**—Constitutional conditions preceding or accompanying the symptoms must be met according to indications. The diet should be designed to aid nutrition to the fullest extent. To increase circulation in the effected limbs, these parts should be kept at absolute rest in an elevated position. When cold, they should be well wrapped in cotton-wool. Massage is sometimes useful. Gangrene must be treated according to sound surgical principles.

Galvanism has been found useful in some cases. The positive pole is applied over the brachial or lumbar plexus according to the extremity to be treated, while a basin of water is made the negative electrode into which the hand or foot is placed.

*Secale*, *arsenicum*, *argentum nitricum*, and *glonoin* are suggested as remedies.

## FACIAL HEMIATROPHY.

Facial hemiatrophy is a peculiar form of wasting of one lateral half of the face, in which the atrophy involves particularly the bones and connective tissues, and dependent upon pathological conditions at present unknown. The etiology of the affection is likewise obscure. It is a matter of clinical observation that the majority of cases have their beginning in childhood. Cases, moreover, occurring at this time present greater differences between the healthy and diseased sides, because in them the effects of deficient growth are added to an actual atrophy. In most cases no cause whatever is assignable. Some few cases follow acute infectious fevers as scarlatina and typhoid fever.

**Symptomatology.**—The characteristic symptom of facial hemiatrophy is a gradual atrophy of one half of the face. The bones of the forehead and face on the affected side become much smaller than those on the healthy. The overlying tissues cover these tightly, and so fit into all depressions. Thus the affected side presents an expression entirely different from the other, as much so as if it belonged to a different individual. The cartilaginous tissues likewise suffer. The eye on the affected side is sunken from involvement of the orbit. The muscles of the face are not paralyzed or atrophied, nor do they exhibit alterations in their electrical reactions. Sensory disturbances appearing in the shape of anæsthesia may be present in any or all of the parts supplied by the fifth nerve. The skin is very much wasted, to the extent even of being one-tenth of the thickness of the normal.

**Diagnosis.**—Differentiation is only required from atrophic palsies limited to the muscles supplied by the fifth nerve and congenital facial asymmetry. In the former the paralysis with reaction of degeneration and the predominance of muscular over osseous atrophy furnish all sufficient distinctions. In congenital asymmetry the difference is never so great as in facial hemiatrophy.

**Prognosis.**—The course of the disease is slowly progressive over a long period of time, when the condition becomes stationary. Life is not endangered.

**Treatment** is utterly unavailing.

## FACIAL HEMI-HYPERTROPHY.

Probably similar in their pathology to facial hemiatrophy are those rare cases of hypertrophy of one half of the face. Of the few cases reported, some have been congenital, while others have been acquired and progressive. No opportunities have been afforded for pathological investigation.



# DRUG HABITS AND INTOXICATIONS.

## ALCOHOLISM.

Alcoholism has so long been discussed from a standpoint of morals, that its consideration from a strictly medical point of view has been greatly hampered. By many, excessive alcoholic indulgence is regarded as a vice during the period in which the alcoholic habit is formed. This position is undoubtedly true in a great measure. There are, however, many individuals in whom the excesses are the direct result of faulty heredity or constitutional peculiarities; and in them alcoholism constitutes a veritable disease. The alcohol habitue may present one of several types. He may be a man addicted to taking large quantities of liquor daily, with or without acute intoxication; or he may be in the habit of "going on periodicals;" *i. e.*, for months, perhaps for years, he remains a total abstainer, and then, without warning, he suddenly begins a wild indulgence in drink. The causes leading up to these periodical excesses have afforded an interesting subject of study. A morbid craving is, of course, the starting point. This craving usually seems to be held completely in abeyance, but sometimes under the influence of excessive mental or physical strain in business, grief, financial losses, etc., it becomes active, and the victim yields to the impulse. In other cases the craving is made active only by indulgence. The taking of a single drink lights up an unquenchable thirst for alcoholic stimulants. Most of these cases possess a neurotic constitution by heredity. Inquiry shows either that one of the parents was an inebriate, or that neurasthenia, epilepsy, neuralgia, insanity, or other neurotic disorders exist in the family. The inebriate may, moreover, transmit to his offspring various neuroses.

Social causes for the production of alcoholism exist. Among the poor, bad hygiene and insufficient food start the craving which ends in a life of drunkenness. Certain occupations expose their followers to more or less constant temptation; notable among them are those in which liquor is constantly about, and such as require frequent "treats" and conviviality.

Alcoholism is brought under the physician's observation in several phases: (1) Acute alcoholic intoxication or drunkenness; (2) chronic alcoholism; (3) delirium tremens; and (4) inebriety.

## ACUTE ALCOHOLISM.

This comes under the physician's care only when it is supposed that some other trouble is present, or when such large quantities of alcohol have been taken as to amount to a veritable poisoning. The symptoms of intoxication are well known to all and require no extended description. The excessive indulgence is shortly followed by mental changes, changes corresponding to the disposition of the individual. The quarrelsome man becomes more so; the generous highly improvident; the quiet man, morose; the sociable, loquacious, etc. Drunkenness brings forth all too plainly its subjects' natural tendencies. The mental changes are associated with motor trouble, the well-known alcoholic inco-ordination. The face is flushed, the conjunctivæ injected, and finally the patient lapses into narcosis. The pulse is now full; respiration becomes deep, but rarely stertorous; the pupils are dilated; and the temperature is subnormal.

Such is the condition presented by the ordinary drunk. Occasionally, owing to personal susceptibility, or to very excessive drinking, more serious symptoms prevail. They come on more rapidly. The preliminary stage of excitement is absent; stupor is an early phenomenon. The face is pale; the pupils are generally dilated, and inactive to light; the pulse is weak; and the skin very cold. Loss of consciousness is profound. Death takes place through paralysis of the respiratory centres. In some cases of alcohol poisoning the patient regains consciousness, and is apparently doing well, when suddenly after moving about in bed, he dies.

Acute alcoholism generally corrects itself within a few hours, if the patient is kept from further indulgence. It is of chief importance to the physician for diagnostic reasons. Often it happens that cases of apoplexy, uræmia, head injuries, etc., are incorrectly pronounced drunken. In ordinary intoxication, the unconsciousness is not so profound as to render rousing of the patient impossible. It will be discovered that in his restless movements, there is no loss of power of any of the limbs. His temperature is subnormal. These data are all sufficient to differentiate intoxication and apoplexy.

From *uræmia*, it is distinguished by the fact that the uræmic coma is rarely of rapid onset, by the diminution or absence of the urinary flow, the presence of dropsy, etc.

In all doubtful cases, it is the best plan to regard the patient as a sick man, and care for him accordingly.

## CHRONIC ALCOHOLISM.

Alcoholic drinks, taken to habitual excess over a term of years, produce disease of various organs of the body, notably, however, of the ner-

vous system, stomach, liver, and kidneys. These effects follow, whether or not the drink has been indulged in to the degree of intoxication.

(1) **NERVOUS SYSTEM.** The brain and spinal cord are especially apt to be affected in chronic alcoholism. The mental condition of the patient undergoes a change in the direction of deterioration of the various faculties. Memory grows poor. The usual mental strength of the patient diminishes. In very advanced cases, it may even amount to a dementia, resembling very closely paralytic dementia. Whether or not excessive indulgence in alcohol can produce general paralysis is a debatable question. Cases resembling this disease very closely have been reported, and to them has been applied the term, alcoholic pseudo-paresis. The mental symptoms from alcoholism cease to progress as soon as the patient's habits are changed. It is rare, however, for an improvement to ensue, if the condition is at all an advanced one. There is nearly always a marked change in the moral tone of the patient. This may go on until there is almost complete abolition of the moral sense.

Of the general nervous symptoms producible by alcohol, tremor is the most constant, and, moreover, one of the earliest to appear. In the beginning, it is observed in the morning only, appearing first in the hands, and subsequently extending to the lower extremities and muscles of articulation. In the legs it is very liable to escape observation. It is always greatly relieved by a drink of whiskey.

Epileptiform convulsions appear in old cases. They are generally a manifestation of organic nervous changes produced by the alcohol. But we have occasionally observed patients with typical epileptic fits, which would only make their appearance during or after a spree.

Many symptoms common to general neurasthenic states are seen. These present no peculiarities in chronic alcoholism.

Lesions of the peripheral nervous system are very frequent. These are included under the head of multiple neuritis, a condition which has already been considered in another section of this work, and to which the reader is now referred. Symptoms from this cause are characterized by their tendency to appear peripherally, and to progress centrally. Many and varied are the morbid sensations experienced. The patient may feel as if myriads of ants or other insects were crawling over him. Diminished sensory functions are not uncommon. Sometimes there is a general hyperæsthesia, the slightest touch causing severe pain. It is most marked in the extremities and lower portion of the abdomen. Severe neuralgic pains in different portions of the body may be experienced.

Sleep is often disturbed. The patient is sleepless, or there are twitchings of the muscles, arousing him from sleep. Dreams are distressing.

It has been said that subacute and chronic myelitis may ensue from excessive alcoholic indulgence. It is more than probable, however, that the cases thus described were really instances of multiple neuritis.



The lesions producing these symptoms are not at all characteristic. Inflammation of the meninges is most frequently found. In chronic cases, a meningo-encephalitis occurs.

(2) DIGESTIVE SYSTEM. Chronic alcoholism is often attended by disorder of the digestive tract. The lesions may affect one or all portions of it. Those most frequently observed are catarrhal conditions of the throat and stomach. The symptoms arising therefrom are in no wise different from those common in the same pathological conditions from other causes. There is sometimes experienced a weak, sinking sensation in the epigastrium in the morning, relieved as soon as the patient takes a drink of whiskey.

The liver is rather frequently diseased, the lesions being either a congestion, a chronic inflammation, or a cirrhosis. The latter affection is commonly regarded as the most characteristic, but recent investigations show that cirrhosis of the liver does not occur among the intemperate to the degree that was at one time believed. There seems to be a predisposition necessary for its production, since it sometimes occurs in those whose indulgences have not been very marked, and is absent in those addicted to most remarkable excesses for years.

The kidneys are often affected in alcoholic habitues, the especial lesion being an interstitial nephritis, which is believed to result from the arterial changes produced by the alcohol.

Other organs are involved in chronic alcoholism; but nothing of note need be said other than what has already been stated in referring to special diseases in other portions of this work.

The treatment of alcoholism is anything but an easy matter. Many remedies have been vaunted, and have alike proven unsuccessful. The nux vomica preparations are undoubtedly the most efficient. To cure the habit, however, requires complete isolation of the patient in an inebriate asylum for a term of months.

For the special symptoms arising from disease of the various organs, remedies and hygienic measures must be recommended according to general indications.

## DELIRIUM TREMENS.

**Synonyms.**—Mania a potu; delirium potatorum.

**Etiology.**—Delirium tremens is a form of delirium occurring only in those addicted to excessive and prolonged indulgence in alcoholic beverages. It very rarely attacks those who, temperate for most of the time, indulge to excess at periods. The exciting causes determining the onset of the disease are found in an habitually heavy drinker either in temporary excess or in the occurrence of some injury, accident, or acute inflammatory disorder. The former of these causes is beyond all doubt the most frequent. In hospital practice one meets with many cases

admitted by reason of some slight accident, in which the symptoms develop within a very few days. I have even known an acute poisoning, as from ammonia or opium, to precipitate an attack. Of the acute inflammatory affections liable to excite the disease, pneumonia is the most common, and, unfortunately, one of the most dangerous. It is a generally accepted opinion among the laity, one, too, widely disseminated among the profession, that delirium tremens is occasioned by the sudden withdrawal of liquor from the habitual toper. To this cause is attributed the frequency of the disease among recently admitted hospital inmates. However strong may be the circumstances pointing to the correctness of this view, it must not be forgotten that there are cogent facts indicating its falsity. One of the earliest symptoms of delirium tremens is disgust for liquor. The patient, no longer craving it, does not indulge. To this sudden change of habit the disease is attributed, rather than to recognize the change as but one of its symptoms. Men are more frequently the subjects of delirium tremens than are women, undoubtedly because they indulge more often, rather than because of any sexual predisposition to this manifestation of alcoholic poisoning. Men of middle life form the majority of the subjects, because of the length of years of indulgence usually required for the production of the disease.

**Symptoms.**—The patient at the outset of the disorder exhibits symptoms common in chronic alcoholic habitues. He is nervous and restless. Tremor becomes prominent. Sleep is greatly disturbed; there may even be insomnia. He dreams, the subjects being mainly of an unpleasant character. They may be so vivid as to impress themselves on him during his waking hours. In very many cases disgust for liquor is manifested; in others there is the reverse condition, a morbid craving for alcohol.

The delirium first shows itself in an undue loquacity. The subjects discussed are handled in a rambling manner. Very often the patient declares himself perfectly well, and demands that he be permitted to go home, if he is in the hospital; or that he may go to work, if he is being treated at his home. Then visual hallucinations appear. These are nearly always of a horrible nature. The patient sees mice, snakes and other repulsive animals about him. He may even see persons or mobs bent on his destruction. Auditory hallucinations, though less common, are frequently present. The patient's whole manner is in keeping with his false ideas. He exhibits an expression of terror or horror; he endeavors to escape from his imaginary enemies, even resorting to violence to accomplish his aims. This violence, however, is one of defence rather than of offence. He may simply try to escape through any avenue available, not hesitating to jump through windows. Sometimes his chief hallucination is that of insects and vermin crawling over him.

In this case, he will be observed to be constantly picking at himself to remove the disgusting objects.

Very often the violent conduct brought on by the hallucinations requires restraint. Usually, however, a little patience on the part of the attendant, and a few firm but gentle words, are all sufficient to quiet the sufferer. While the patient lies in bed, he picks restlessly at the bed-clothes, the movements being characterized by a very marked tremor. The symptoms are always greatly aggravated at night. The delirium usually continues for from two to five days, when it subsides, and the patient slowly regains strength. In less fortunate cases, a condition of exhaustion sets in, the pulse becoming weak and rapid. The most frequent cause of death is heart-failure. Some cases end in coma or convulsions. Ofttimes the extreme prostration in the fatal cases is associated with all the symptoms common to typhoid conditions generally.

Fever is present in the majority of cases of delirium tremens, the temperature however rarely rising above  $102^{\circ}$  F. or  $103^{\circ}$  F. Appetite is as a rule very bad, it being only with the greatest difficulty, oftentimes, that the patient can be induced to eat. The pulse is increased in frequency, ranging from 110 to 150 per minute. It is not unusual to find the urine albuminous, even though no kidney disease be present. It is more common, however, to find the disease engrafted on a constitution already undermined by chronic kidney disease, in which case the urinary condition is greatly aggravated by the delirium tremens.

**Complications.**—It is not at all uncommon, as already intimated, to find delirium tremens associated with traumatisms, both accidental and operative. It also complicates certain acute diseases, notably, as already stated, pneumonia. Pneumonia may also set in as a secondary disorder, and add greatly to the patient's danger. Its onset may be so insidious as to escape attention unless careful physical examination of the chest be made daily. Erysipelas is another not uncommon complication.

Syphilis, especially the tertiary cerebral variety, is often a coincidence in this affection, and the symptoms of these lesions may modify or prolong those due to alcohol.

**Pathology and Morbid Anatomy.**—The changes found after death consist mainly of congestion of the cerebral cortex. Other structural alterations are dependent mainly upon the long-continued use of alcohol, and include chronic or acute meningitis, and the well-known effects of alcohol on the viscera generally.

**Diagnosis.**—As a rule there is no difficulty in the differentiation of delirium tremens from the conditions which simulate it. The most frequent diagnostic error lies in the failure to recognize the existence of other disorders, such as chronic nephritis or pneumonia. The peculiar character of the hallucinations, though not by any means pathognomonic,



should serve in a measure to guide to a correct decision, especially in the presence of a history of long-continued alcoholism, and the absence of symptoms indicative of meningitis, acute mania, or paralytic dementia, which are the disorders most likely to simulate delirium tremens symptomatically. The mental state of the last-named is very different from that of the disease under consideration.

**Prognosis.**—This is, as a rule, favorable in first attacks of the disease occurring in individuals whose viscera have not undergone serious organic changes by reason of the long-continued alcoholic indulgence. Hospital cases do not offer as good an outlook as those seen in private practice, undoubtedly because of the impoverished condition of the patients. Statistics from the most reliable sources show an average mortality of about 10 per cent. Old age makes the prognosis more hopeless. Of the visceral complications, pneumonia and chronic Bright's disease are the most unfavorable.

**Treatment.**—The most important point in the treatment of delirium tremens is careful intelligent nursing. All undue excitement of every kind should be avoided. No one should be admitted to the sick-room but the nurses and the physicians. The room should be quiet and the light in it greatly subdued. In some instances the latter precaution serves only to intensify the patient's hallucinations, in which case, of course, it should not be adopted. The extent to which the patient should be physically restrained requires nice judgment. In many instances the firm bonds only add to the patient's furor. The greater the experience of nurse and physician in the management of these cases, the less likely is physical restraint of any kind to be found necessary. Very often indeed a few judicious words will cause a violent patient to lie down. When restraint is required, it should be of as mild a nature as possible. Probably the best means is that of holding the patient in bed by a sheet thrown over him, and fastened at the sides of the bed. Some few cases remain uncontrollable by mild measures. With them, the strait-jacket or leather handcuffs must be brought into requisition.

The great desire on the part of delirium tremens patients to escape necessitates careful watching. Windows must be securely fastened. Indeed, it is exceedingly unwise to care for such a case excepting on the first floor of a dwelling, unless the windows be protected by iron bars and efficient attendants are constantly on hand.

Hydro-therapeutic measures are sometimes useful. A warm wet pack often acts as a very efficient sedative.

The diet must be nutritious. It should consist of milk, eggs, and well-made beef tea. The patient should be fed every three hours. It is well to season the beef tea with cayenne pepper, or add to each cupful, from fifteen to thirty drops of tincture of capsicum.

The advent of sleep is always to be regarded as a most favorable

symptom. For this reason it has been urged that hypnotics be administered freely in order to bring about this condition artificially. The employment of ordinary doses of bromide of potassium and chloral, the drugs usually recommended in these cases, fails utterly to produce sleep. One may make an even more positive statement and say that doses of bromide and chloral capable of producing sleep in a severe case of delirium tremens are so large as to greatly weaken cardiac action. The exhibition of small doses of bromide or chloral is silly because useless; large doses are dangerous. Therefore both drugs should be expunged from the therapy of delirium tremens. If hypnotic treatment is necessary, one drug only should be resorted to, and that is the hydrobromate of hyoscyne. It should under no circumstances be administered in doses of greater than one one-hundredth of a grain, and at intervals shorter than four hours. The best effects are obtainable from its hypodermic administration.

To obviate cardiac failure, heart stimulants are sometimes necessary. These include such drugs as coffee, caffeine, cocaine, and aromatic spirits of ammonia. Alcohol will sometimes be found necessary in this connection.

The best remedy in these cases is *strychninum sulph.* 2x, which should be administered in one grain tablets at intervals of from four to six hours.

When the temperature is high, the cold douche or a cold wet pack should be employed.

As to remedies, *hyoscyamus* is as a rule better adapted to the type of delirium present than is any other remedy. Occasionally, however, *belladonna* or *stramonium* will be more suitable, while in cases in which asthenia is profound, *lachesis* or *arsenicum* will prove useful. *Tartar emetic* is adapted to cases in which the gastric symptoms, especially the vomiting, are prominent. It is all the more indicated when pulmonary complications are present. *Arsenicum* is indicated by symptoms of gastro-intestinal irritation and albuminuria. Indeed with renal symptoms prominent, no remedy is as likely to prove beneficial as is arsenic. *Cannabis indica* and *actea racemosa* are indicated by the character of the patient's hallucinations. With the former remedy, objects appear larger in size than normal, and seem at an unnatural distance; minutes appear like hours. Under *actea racemosa*, the patient sees rats, mice and other unpleasant objects; he talks incessantly, not keeping himself on any one subject long enough to give any connected idea; he has occipital headache.

When suppression of urine threatens, hot fomentations should be applied to the loins, and hot foot-baths given.

## MORPHINISM.

Although opium has been known since remote antiquity, addiction to its use as a vice or habit is of comparatively recent date. The opium habit seems to have started in Persia and Turkey during the Middle Ages. It was introduced into India in 1511, and into China in 1644. Its use in the latter country did not become general until 1767, in which year the East India Company began its importations of opium into China. The habit became such a national vice that laws looking to the suppression of the use of opium were enacted, and severe penalties, including confiscation of the victim's property, and even death, inflicted. These stringent measures were unavailing. The opium habit found a foothold in the United States about a half century ago. The habit of smoking opium was brought into this country, in 1868, by the Chinese, and speedily found many victims along the Pacific coast. At the present time there are all over the country establishments known as opium joints, carried on for the most part by Chinese, where this vice is practised.

**Etiology.**—The majority of cases of morphinism or the opium habit find their origin in the excessive use of morphine in the treatment of painful affections, especially those of a chronic nature. The hypodermic use of the drug seems especially to be attended by this danger. The majority of cases coming under my notice have arisen from the hypodermic use of morphia for the relief of severe headaches and other forms of neuralgia. Probably one-fourth of the cases begin the habit for sensuous reasons only. This latter is the main cause of morphinism in Oriental countries.

**Pathology and Morbid Anatomy.**—The anatomical changes resulting from morphinism are not well understood. In a general way it may be stated that they are secondary to the effects of the drug on the glandular system and general nutrition. The secretory functions of all the glands are held in abeyance. The changes in the nervous system are finally degenerative in character.

**Symptoms.**—The first effects of a dose of morphia are those of a cerebral stimulant. As these phenomena pass off depression sets in. In the beginning of the habit but a small dose of the drug is required to produce the desired effect. As the habit continues this dose increases, until quantities poisonous to the ordinary individual are taken. There is, however, a limit in dosage beyond which the patient cannot and does not go, and this limit varies according to the idiosyncrasy of the indi-



vidual. Some reach it at from two to five grains daily ; others not until they are taking as much as a drachm of morphia. It is very rarely that these patients take an overdose of the drug. Instinct seems to preserve them from this mishap. The dose taken by opium smokers ranges from six grains in the beginning to over three hundred grains daily when the habit is confirmed.

Symptoms of the morphia habit itself usually appear in from two to six months after it is formed. Even here this time varies greatly according to personal susceptibility and national temperament. Oriental nations bear with the habit far better than Americans and Englishmen. There is even strong evidence to show that the average opium eater of India is not even harmed by this habit. The first condition observed is the craving for the drug. As soon as the effects of the dose wear off, the patient becomes nervous and restless, breaks out into a perspiration, experiences nausea and becomes fatigued. The general symptoms are the result of the effects of opium on the secretory glands of the stomach and intestines. Naturally indigestion ensues, and with it general malnutrition. The patient emaciates and the complexion assumes a sallow hue. The skin exhibits a chronic flushing and even purpuric spots. Levinstein has described a fever of malarial type resulting from the morphia habit. The temperature may rise to 104° F. Quinine does not have any effect on it. A dose of morphia relieves it promptly. The fever is very apt to be accompanied by neuralgic pains and intense prostration.

The influence of the opium habit on the mind is marked. A condition resembling very closely that of delirium tremens ensues on the sudden withdrawal of the drug. It differs from the alcohol delirium in its shorter duration, generally disappearing spontaneously in from twenty-four to forty-eight hours, and the prompt relief obtained from a dose of morphia. In the majority of cases of morphinomaniacs, there is a general mental deterioration. Moral sense seems to be completely obtunded. These patients are proverbial liars. One cannot place the slightest dependence on any statements they may make. They are likewise malicious in disposition. In many cases there is a steady deterioration of all the mental faculties.

In the absence of an acknowledgment of the cause of the ailment on the part of the patient a diagnosis is a very difficult matter. It is ordinarily recommended that the discovery of morphia in the urine will settle the question. This is true. But the tests enabling us to make this discovery are of a very elaborate character, requiring an expert chemist to employ them successfully. Then, too, morphia is not always to be found in the urine even in confirmed habitues. For these tests the reader is referred to von Jaksch's "Clinical Diagnosis," Taylor's "Treatise on Poisons," or Wormley's "Microchemistry of Poisons."

**Prognosis.**—The prognosis of morphinism is not very favorable.

Very many cases relapse after discharge from the asylums. The outlook is greatly influenced by the length of time the habit has been practised, those who have used the drug for many years being practically incurable. Cases which began the habit for the relief of some painful affection are unfavorable, if the primary disease still exists. The existence of a strong neurotic heredity makes success difficult.

Even with success temporarily obtained, these patients are in the greatest danger of a relapse, for the ingestion of the smallest portion of the drug is sufficient to awaken the old morbid craving.

**Treatment.**—Several methods of treatment have been advocated. The best is probably that advocated by Erlenmeyer, and in which the drug is withdrawn rapidly, that is within a period of from five to ten days. The slow method is advocated by many, but is objectionable because of the length of time the patient is kept under treatment, and the prolongation of craving occasioned. The sudden and complete withdrawal of all morphia has been advocated. This procedure is almost certain to be followed by furious delirium and prostration, requiring great care in their management. These dangers escaped, the rapid method is undoubtedly a good one, but the patient must be in a good physical condition to tolerate it. In Erlenmeyer's method the diminution in dosage is graduated according to the case. No hard and fast rule for guidance can be established. Whatever plan of treatment be adopted, the patient must be completely isolated from home and friends. No chance for success exists otherwise. It is astonishing to note the extent to which these sufferers will go in order to procure morphia. Even in a sanitarium, it is absolutely necessary to scrutinize closely every communication with the outside world. Even the servants in attendance must be watched lest they be bribed.

During the early days of the treatment the patient must be kept in bed. The diet should be highly nutritious, and consist of milk, eggs, meats, etc.

The delirium and intense prostration which often mark the early days of treatment, may be combatted by judicious doses of morphia. But the drug should only be administered if these symptoms become a serious matter.

Diarrhœa not infrequently occurs, for which *china*  $\phi$  is the remedy.

The patient must be kept under surveillance for several weeks after the drug has been abandoned; otherwise a relapse is almost certain to follow.

Other methods of treatment have been advocated, but lack the sanction of authority and general experience. One of these consists in gradually substituting codeia for morphia, and then wean the patient from his codeia. Another consists in withdrawing the drug suddenly, and stimulating the patient with *sparteine* and *nitro-glycerin*.

## COCAINISM.

The existence of the cocaine habit has been called in question by several prominent authorities. The observations of Erlenmeyer and Mattison, however, prove conclusively that it is a form of drug addiction far more dangerous in its results than morphinism or alcoholism. For many years the leaves of the erythroxyton coca have been chewed by the South American natives, it being claimed that their use enabled them to withstand fatigue with a minimum of food and drink. Writers disagreed as to the merits of the plant thus used. It was generally believed that the chewing of coca leaves did not result in a drug habit. When cocaine came into general use as a local anæsthetic, it was employed as a remedy for the opium habit. It was proven that the morphia habitue could abandon his favorite drug while taking cocaine. It was not long, however, before it was discovered that the cocaine habit was even worse in its effects than that of morphia. Very few cases of cocainism occur aside from those arising from injudicious attempts to cure morphinism. Sometimes the habit is started by taking cocaine as a cerebral stimulant, or by its application to the nose in acute rhinitis.

**Symptomatology.**—The effects of cocaine on the circulation are among the first to follow its administration. The heart-beats increase to from 100 to 130 beats per minute, and weaken quite perceptibly from slight efforts. Vaso-motor and respiratory disturbances of various kinds assert themselves. There is a cold, clammy sweat on the extremities, while the skin of the body is dry and harsh. Very early in the habit emaciation appears and progresses to a marked degree. This condition is believed to result from two causes: In the first place, cocaine produces a nearly complete destruction of appetite, so that the patient takes little or no food, and in the second place, it is thought to be dependent upon the deranged glandular action produced by the drug.

On the nervous system, cocaine at first produces an over-activity. There follows a peculiar restlessness and insomnia. When the patient does succeed in gaining sleep, it is accompanied by horrible dreams. The eyeballs have a brilliant appearance, and the pupils are dilated. Hallucinations, delusions, and illusions of various sorts appear. Hyper-æsthesia to touch takes place. There may even be tactile hallucinations, this condition being very characteristic of the cocaine habit. The patient imagines that he feels a bug or a piece of glass beneath the skin, and will spend hours in securing its removal. Finally there is general



brain demoralization. The patient is insane, and has delusions of persecution. Thus he becomes dangerous to his family and neighbors; removal to an asylum may be necessary.

When the habit does not advance to a delusional insanity, the patient exhibits a remarkable loquacity, talking disconnectedly on subject after subject. Ideation and intellection become weakened and make the picture of cocaineism even more characteristic. The patient's habits change. From being tidy and precise, he becomes absolutely filthy and careless. All moral sense is lost. He becomes untruthful, and manifests a degree of malice and selfishness that would put the inebriate to shame.

**Prognosis.**—Fortunately since the recognition of cocaineism, and the abandonment of the cocaine treatment of morphinism, the cocaine habit is rarely met. The acute cure, that is the securing of temporary abstinence from the drug, is not difficult. As soon as the patient is taken away from strict supervision, relapse is very prone to occur.

**Treatment.**—*This necessitates removal of the patient to a properly conducted sanitarium*, where he should be kept for a period of several months. Removal of the drug is not usually followed by as serious results as in the case of morphia. Symptoms arising from this cause, call for treatment according to indications.

## GRAIN POISONING.

### (A) ERGOTISM.

**Synonyms.**—Ignis sacer; ignis St. Antonii; raphania.

**Definition.**—Ergotism is a form of intoxication arising from the ingestion of certain cereals, especially rye, which have been acted upon by the fungoid growth, *claviceps purpurea*, and characterized by gastro-intestinal, gangrenous, or cerebro-spinal symptoms, according to the type of the case.

**Etiology.**—Ergotism, at one time very common in epidemic form, is now mostly limited to certain regions in France, Germany, Russia, and Sweden. Very limited epidemics have occurred in America. The fungoid growth producing the poisoning affects man only when infesting rye. It is especially liable to flourish when a rainy spring is followed by a dry hot summer. The weak and delicate, and those who have had previous attacks, succumb more readily to the action of the poison.

It is customary to study ergotism as of two varieties, viz., the gangrenous and the convulsive. It is believed that the one or the other of these prevails in a given epidemic according as the ergot is richer in one or the other of its active principles, cornutin and sphacelinic acid.

**Symptomatology.**—Acute poisoning by ergot excites numerous gastro-intestinal symptoms, presenting few characteristic features. Should the victim happen to be a pregnant woman, she is almost certain to abort.

In chronic poisoning the first influence is observed on the circulation. The pulse becomes small and tense, and the skin cool. Gangrene of either the dry or the moist variety may next develop.

The spasmodic or convulsive form of ergotism develops by the appearance of quite a variety of nervous symptoms of indefinite character closely resembling those of neurasthenia. They consist of numbness and tingling, mental and physical depression, vertigo, change of disposition, etc. Then come the true convulsive phenomena. They consist of either clonic or tonic spasm, and are associated with preservation of consciousness. The muscles of the upper extremity, notably those of the forearm and hand, are especially apt to be involved. Sometimes the seizures resemble very closely those of catalepsy or epilepsy, in the latter case often being followed by a characteristic post-epileptoid state. Even atrophic paralyses have been known to ensue upon ergotism.

The sensory symptoms of ergotism consist of morbid sensations in great variety.

Of late years, the ataxic symptoms produced by ergot have excited great attention. Tuzcek described an epidemic occurring about Marburg, in which many of the victims suffered from symptoms resembling closely those of typical tabes dorsalis. There were ataxic gait, diminished knee-jerk, lightning-like pains, occipital headache, etc.

**Prognosis.**—The majority of cases recover as soon as the patient is removed from the unfavorable influence. When gangrene or severe exhaustion has been produced, there is danger of death from cardiac collapse. Recovery from the ataxic and cerebral symptoms is necessarily slow. Death may result from these.

**Treatment.**—This resolves itself into a discovery of the cause of the trouble and the prompt removal of the same. Then symptoms and conditions should be treated on general principles. *Camphor, opium, arsenicum, solanum nigrum, belladonna, hyoscyamus, and stramonium* are the principal antidotes.

### (B) LATHYRISM.

This is poisoning from the seeds of a leguminous plant, the lathyrus, two varieties of which are active, the *lathyrus cicera* and *lathyrus sativa*. The poisoning generally arises from the admixture of the powdered seeds with flour in the preparation of bread. So far as is known, this occurs only in the countries of Europe and Africa bordering on the Mediterranean Sea.

The symptoms produced by the lathyrus are those of spastic paraplegia, which may increase to complete helplessness. The bladder is apt to be involved, there being either retention or incontinence of urine. Muscular atrophy rarely occurs.

The pathological condition causing the symptoms seems to have been undetermined. Some consider it a slow sclerosis; but against this idea is the fact that most cases make good recoveries when removed from the deleterious influence. Others regard it as a transverse myelitis, with secondary degeneration of the lateral columns of the cord.

### (C) PELLAGRA.

Pellagra is a disease probably caused by an organic virus, or its product, taken into the system with spoiled maize. Some authorities regard privation and want as entering into the etiology of the trouble. It occurs only in France, Italy, Spain, Poland, Algeria and portions of Austria. It is entirely unknown in Germany, America, England and Belgium.

The symptoms may be divided into three classes: (1) Cutaneous; (2) gastro-intestinal; and (3) cerebro-spinal. The cutaneous symptoms consist of an erythema, which attacks principally uncovered portions of the body. The affected area of the skin is bright-red in color, and is the



seat of considerable burning and itching. Sometimes papules and vesicles appear. Later, desquamation takes place.

The digestive symptoms consist of dyspepsia and diarrhœa, especially characterized by an association with cracking and fissuring of the lips, sponginess of the gums, aphthous sore mouth and cracking of the tongue. Diarrhœa is a prominent symptom, and is very exhausting.

The spinal cord is severely affected, symptoms closely akin to those of ataxic paraplegia presenting. They are probably the result of a degeneration of the lateral and posterior columns produced by the poison, but differing from the idiopathic affection by a not infrequent involvement of the anterior cornua and chronic inflammation of the pia mater.

Sometimes the brain becomes affected. The mental powers wane; vertigo, tremor, irregular movements, hallucinations of sight and hearing, slow speech and incoherent ideas prevail. Paralysis of the pharynx has been reported.

**The Prognosis** depends largely upon the previous hygienic condition of the patients and the length of time of exposure to the poison.

**The Treatment** is entirely hygienic and symptomatic.

## PTOMAINE POISONING.

The term ptomaines includes a number of substances similar in chemical composition and physiological action to the vegetable alkaloids, but differing from them in that they are the products of putrefactive changes taking place in dead animal tissues or secretions or their products.

**History.**—Although the fact that putrefying matter was capable of producing very serious effects and even death when taken into the healthy organism, has been well known for many years, the knowledge of the existence of ptomaines as such is of comparatively recent date. Selmi, in 1875, was the first to formally direct scientific attention to this subject. In previous years many theories to account for the severe illnesses following the ingestion of certain articles of food not ordinarily poisonous, had been presented. Some authorities believed the symptoms to result from the admixture of a virulent mineral poison with the food. Some were convinced that hydrocyanic acid had been formed in the natural changes of decomposition. Many thought that the meat used was taken from diseased animals. Still others attributed the deleterious effects to poisonous plants mixed with the meat during cooking, in mistake for innocuous herbs. The first to offer an opinion approaching at all near to the truth was Panum, who, in 1856, extracted from the cadaver a ptomaine in an impure state. In 1868, Bergmann and Schniedemann discovered sepsin, and in 1869 Zuelzer and Sonnenschein obtained from a cadaver a substance which exhibited physiological effects similar to those of atropia.

Since Selmi's day the investigation of the ptomaines has been carried actively on. A large number of these substances has been discovered, and their properties investigated. They have been obtained from numerous sources, from putrefying flesh, from cheese and other milk products, from putrid gelatin and from poisonous fish, notably among the latter from mussels. They are even believed to exist in the normal living body, being there found in the urine and in the products of digestion. The bacteria occasioning certain infectious diseases are believed to produce ptomaines of their own, a class known generally as the pathogenic ptomaines.

The relationship between ptomaine poisoning and bacterial infection must, in the present state of knowledge, be looked upon as an unsettled one. Undoubtedly in some cases it is the former, and in others the latter agency that lies at the foundation of the symptoms. Some-

times the tainted meat loses its poisonous properties by thorough cooking; in other cases it does not; indeed, it may happen that the poisonous alkaloid may be generated in the very act of preparing the food. The ptomaines themselves are often unaffected by the action of the heat. Frequently they are of such unstable composition as to be destroyed by the chemical processes employed for their recognition. Very slight circumstances oftentimes determine their existence in putrefying matter. Some are present in one stage of the putrefactive process; others only at another. Their nature is largely influenced by the character of the albuminoid or gelatinoid undergoing decay, and the degree of moisture and heat to which it is subjected, and whether or not while these changes are going on, it is exposed to atmospheric oxygen.

Reference has been made above to the similarities existing between the ptomaines and the vegetable alkaloids. In all cases save one, there are means by which a ptomaine may be detected from the vegetable alkaloid it most resembles. That exception is muscarin, which, in chemical composition and physiological action, is apparently identical with the alkaloid of *agaricus muscarius*. This is an important matter from a medico-legal standpoint, for cases have arisen in which chemists have been deluded into the belief that they had detected the presence of morphia and coniine, when in reality the reactions, apparently disclosing these poisons, depended upon the ptomaines.

The data by which tainted food may be recognized are at present very unsatisfactory. Fortunately, ptomaine poisoning is rare, so that the necessity for this recognition is seldom of importance. The ptomaines occur for the most part in the early stages of putrefaction, even before any changes perceptible to the senses are noticeable.

Meat poisoning may occur from the ingestion of beef, veal, mutton, sausage, and ham. The symptoms may be described in a general way as those of a violent gastro-intestinal irritant. They come on usually within twelve hours from the time of taking the tainted meat, although their advent may be delayed until two days or longer. Usually they appear suddenly; exceptionally they may be preceded by a so-called prodromic stage characterized by languor, loss of appetite, nausea, rigors, vertigo, faintness, cold sweat, tremor, etc. The most frequently observed initial symptom is a sense of chilliness. Sometimes there is headache or pain in some other portion of the body. Then appear abdominal pain, diarrhœa and vomiting. The pain is very severe and is commonly accompanied by cold sweat. The stools are exceedingly offensive in odor and dark in color. Prostration is profound. The skin is usually cold in the early stages; later the temperature rises, and the thermometer may indicate hyperpyrexia. Spasmodic symptoms, such as accompany abdominal irritation, as cramps in the calves, spasms of the different portions of the body, muscular twitchings, may be exceptionally



present. Sometimes there are pains in various joints, sensations of numbness and formication in different parts, drowsiness, hallucinations, and visual disturbances. In fatal cases, collapse precedes the final stage.

Sausage poisoning is also spoken of as *botulism* or *allantiasis*. It is very rarely observed at the present time. It was at one time quite common in the Swabian regions of Germany. This condition is remarkable for the length of time over which the symptoms may persist. They indicate a most profound affection of the entire system. Gastro-intestinal irritation is present, but not to such a degree as to render it liable to be mistaken for gastro-enteritis. The sausages producing the mischief are generally blood or liver sausages. The poison does not seem to be equally distributed throughout the mass, the interior portions especially being the most deleterious. On cutting across the poisonous sausage the section will have a dirty-greenish color, and a cheesy-like smeary consistence. The odor is said to resemble that of putrid cheese. Symptoms may appear in from one to nine days after partaking of the poison. The first signs are usually general discomfort and nausea, or others already mentioned in the previous paragraph as resulting from meat poisoning. The gastro-intestinal symptoms may last for several days, when those of a nervous character appear. The patient then has headache, vertigo, and a general apathetic condition. Extreme muscular weakness is characteristic. Visual power becomes diminished. The patient sees as if through a cloud or mist. Certain muscles of the eye become paralyzed; thus there occur diplopia and ptosis. The pupils are dilated. Exceptionally total blindness has occurred. The parts to which the glosso-pharyngeal nerves are distributed exhibit disturbances of function. At first there is simple difficulty in swallowing. This may be increased to absolute inability to swallow. The tongue is likewise affected, thus interfering materially with speech. The mucous membranes generally have their secretions lessened. Thus we note dry mouth, and constipation. As is to be expected, the heart is weak in its action. Favorable cases are marked by a slow convalescence. Fatal ones end within from a few hours to three weeks.

Quite a variety of milk products have been found to unexpectedly poison families and communities. Prominent among the articles possible of undergoing poisonous changes are cheese, ice-cream, cream puffs, and custards. The precise nature of the poison generated is not at all understood. Sometimes it seems to have been engendered during the process of cooking, for other foods made from precisely the same materials have not proven deleterious. In the case of ice-cream, it is believed by many that the poisonous agent is of mineral nature derived from the can. Dr. Geo. S. Hull has offered a very plausible explanation of ice-cream poisoning. As he says, the can, the contained cream, and the galvanized iron paddle give all the requisites of a galvanic cell. If the

paddle is permitted to remain in the cream after churning is completed, some of the metal will be dissolved and contaminate the cream. Other cases of poisoning must arise, however, from the generation of a ptomaine. The symptoms are those of a violent gastro-intestinal irritant.

Poisoning from fish is about as mysterious as other varieties of ptomaine poisoning. Naturalists and others have told wonderful stories of fish, the partaking of which brought sickness and even death. Whether or not there is such a thing as a fish the flesh of which is always, that is at all times and under all circumstances, poisonous is not definitely known. Probably the poison quality is often derived from the food. This is well illustrated in the case of the mussels in Wilhelms-haven Bay. Non-poisonous mussels transplanted to these waters speedily became poisonous; while those which had been there lost their dangerous property after removal to another locality.

The symptoms from poisonous fish are of a choleraic character. Mussel poisoning is accompanied by profound nervous disturbance without gastro-intestinal irritation. The symptoms are those of a rapid collapse, death taking place sometimes within two or three hours.

The treatment should be directed first to the removal of the poisonous food from the stomach, following which symptomatic indications should be the guide.

Hering recommended in sausage and meat poisoning the use of a beverage of vinegar and water taken in large quantities. At the desire of the patient, these acids may be alternated with black coffee or black tea freshly prepared. For the dry throat and gastro-enteric symptoms, Hering considered *bryonia* the most important remedy, and for the symptoms remaining after this medicine had accomplished its work, *phosphoric acid*, *arsenicum*, or *creasote*.

The symptoms arising from the ingestion of poisonous fishes are best met by the administration of *arsenicum*, *belladonna* or *capsicum*.

Ice-cream poisoning, and indeed toxic symptoms arising from the ingestion of contaminated milk products generally, should be treated by the administration of remedies adapted to gastro-intestinal irritation, as *arsenicum*, *nux vomica*, etc.

## ARSENICAL POISONING.

Acute poisoning by arsenic presents a characteristic picture of gastro-intestinal irritation very readily recognized in practice. Prolonged exposure to the influence of the poison may give rise to a number of symptoms the recognition of which is not so easy. It is of cases arising from this cause that the present article proposes to treat. Arsenic enters very largely into the composition of certain coloring matters, especially green and red. These pigments have been widely used in the coloring of wall-papers, artificial flowers, various fabrics, etc. Certain persons when possessed of an idiosyncrasy to the drug, when exposed to the emanations from these articles, have suffered from symptoms to be shortly described. Cases of poisoning have likewise arisen in those exposed to arsenical fumes in certain manufacturing works. Arsenic in the form of Fowler's solution is largely used, often in extreme doses, by the dominant school of medicine. Sometimes slight poisoning symptoms arise. These consist of oedema of the eyelids, nausea, vomiting, and cutaneous hyperæmia. Pigmentation of the skin has arisen from this cause in a number of instances.

The first symptoms of chronic arsenical poisoning are those referred to the respiratory mucous membrane, and probably are the direct result of local action. They consist of coryza, pharyngeal irritation, and dry cough. The conjunctiva becomes injected; there are also dread of light, nausea, and slight gastro-intestinal disturbance.

Exposure to the poison being continued, quite a variety of annoying conditions are induced. Thus there may appear headache, insomnia, mental and physical depression, tremor, convulsions, faintings, or paralysis. It must not be inferred that these symptoms are all present. It usually happens that but one or two of them constitute the entire source of suffering, and cause the physician considerable trouble in determining their origin.

The treatment consists in the discovery and removal of the cause, and the administration of symptomatic antidotes.



## LEAD POISONING.

**Synonyms.**—Plumbism; saturnism.

**Etiology.**—Of the various forms of accidental chronic metallic poisoning, that by lead is unquestionably the most frequent. Ofttimes the symptoms resulting are so obscure and the *possibility* of poisoning so unsuspected, that physicians themselves experienced in the observation of the malady, have long suffered from it without once entertaining a thought as to the true nature of their ailments. As is the case with other forms of poisoning and disease generally, constitutional predisposition to lead poisoning varies greatly. Some persons succumb to its influence within a few weeks after taking up the deleterious occupation; others work on for years without the slightest suffering. Women are more susceptible to the influence of lead than are men. This statement, recently established by Oliver, is in opposition to previous beliefs. Especially liable to poisoning are those weak delicate women who obtain insufficient food and clothing, and whose lives are lives of toil and misfortune. Persons of a gouty diathesis, when exposed to lead, suffer greatly from its effects. Of all predisposing influences, alcoholic indulgence is the most prolific. Indeed, one need not expect to benefit the sufferer from plumbism unless he abandon any bad habit he may have in this direction.

The sources from which the lead producing the mischief is derived have been described under two headings, the industrial and the accidental. Under the former are included those cases arising from exposure to lead in certain trades, notably white-lead manufacturers, painters, printers, potters, leather cutters, lead smelters, etc. Miners of lead do not suffer. The actual rate of lead poisoning in these various occupations cannot be accurately determined, for many persons suffering from the baneful effects of the metal do not exhibit the characteristic toxic effects, but develop instead chronic degenerative changes in the nervous system and the kidneys.

The most common accidental cause of plumbism is the presence of lead in drinking water. The metal is generally introduced by reason of the water passing through lead pipes, or being permitted to stand in leaden receptacles. Pure or spring water, or water containing vegetable acids, is especially dangerous. Most cases arising from poisoned water supply occur in epidemics.

Cases have also arisen from the contamination of wines by lead salts placed therein by the manufacturers. Poisoning has occurred from the drinking of carbonated mineral waters sold in siphons, the tops of which

are largely composed of lead. Flour ground between mill-stones, crevices in which had been filled up with lead, has been the cause of mischief in some few cases.

Personal causes arising within individuals are notable. The use of hair dyes, face enamels, false teeth set on cheap plates, etc., have exceptionally produced lead poisoning. Poisoning from face powders and enamels is less frequent than is generally believed, probably because the fatty substance used in connection therewith effectually stops up the pores of the skin, and lessens the absorptive powers of that organ.

The avenues through which lead enters the body are the gastrointestinal tract, the respiratory system, and the skin. Much may be done in the way of prevention by personal cleanliness in the trades exposing their workmen to lead. The mere suggestion of this possibility is sufficient for all practical purposes. The particular carelessness in each case must be learned by careful examination of the patients.

Poisoning from lead used as an adulterant is very rare. An epidemic of plumbism occurring in Philadelphia several years ago, arose from the use of chromate of lead by certain confectioners, for the purpose of giving the cakes a yellow color, as if they contained an abundance of eggs. Exceptionally very mild exposure has produced poisoning. Examples of this are found in cases arising from sleeping in freshly painted rooms, or from the drinking of water ordinarily free, but contaminated by reason of having stood in the pipes an unusual length of time.

**Pathology and Morbid Anatomy.**—Chronic lead poisoning leads to anatomical alterations in a number of organs, notably in the nervous system, kidneys and liver. These changes are perceptible by the microscope only, excepting in the cases in which lead is deposited as an insoluble sulphide. Then it appears as minute black spots. This appearance is found most frequently in the intestines.

The ravages of lead on the nervous system are found principally in the peripheral nerves, and especially in the intramuscular nerve twigs. The changes become less and less marked the further the nerve fibres are removed from the periphery. The nerves especially liable to involvement are the radials and their branches. The anterior spinal roots sometimes present evidence of degeneration similar in character to that found in the peripheral nerves. The posterior roots are invariably normal. In some few cases, the anterior cornua of the cord are affected. Some authorities are inclined to believe the spinal cord very frequently affected, basing their opinion on the association of the paralyzed muscles, the supinator longus nearly always escaping. The normal condition of the latter muscle is explainable by the fact that it is the peripheral extremities of nerves that are affected in lead poisoning; and that the radial nerve is not often involved to as high a point as that at which the branch to the supinator longus is given off.

The brain presents no characteristic changes, even in cases in which delirium and other symptoms have been marked.

The liver and kidneys show increase in the formation of connective tissue. Vascular changes are sometimes associated with the renal disease.

**Symptomatology.**—The symptoms of chronic lead poisoning may be epitomized as consisting of the following: (1) anæmia; (2) blue line on the gums; (3) colic; (4) disturbance of the sexual functions; (5) neuralgic pains; (6) various forms of paralysis, mostly of atrophic type; (7) tremor; (8) lead encephalopathy; (9) disturbances of the cranial nerves; (10) renal disease, with its secondary arterial changes; (11) gout.

Of these symptoms anæmia is the first to make its appearance. Very few cases of lead poisoning develop to any extent without exhibiting it. The number of red blood corpuscles is reduced to from 2,500,000 to 4,000,000 per cubic millimetre, and the hæmoglobin to 50 per cent. of the normal. It is currently believed that the effect of lead on the blood is due to its action on the bones, it having been demonstrated that the metal is deposited in this tissue, and by Raimondi that atrophy and degeneration of the bone marrow is a condition attendant upon saturnism. The anæmia naturally leads to general nutritional disturbances, with its consequent prostration.

The existence of the blue line along the gums is well known as a symptom of lead poisoning. Still its presence should not be regarded as absolute evidence of that condition, nor its absence as proof that the patient's illness is not due to lead. The line may be present long after all symptoms of poisoning have disappeared, but how long has not been definitely determined. Oliver holds that it may be expected to disappear not later than three months after the last exposure to the poison. Gowers makes mention of a case where it persisted with but little change two years after the entrance of lead into the system had been stopped. Two forms of lead line on the gums must be recognized. One of these occurs on the margin between the gums and the teeth. It occurs very shortly after exposure to lead-dust, and consists of deposits of that substance altered by the sulphur in the secretions of the mouth. It is therefore a deposit on the gums, and is removable at once by the tooth-brush. The other lead line consists of a narrow bluish-black line on the gums close to the teeth. It occurs only when the gum has receded from the teeth. The gum is apt to be more or less ulcerated and irregular in outline. The teeth are likely to be covered with a brownish pulpy fur. When they are kept scrupulously clean and the gums are in perfect apposition with the teeth, this blue line is absent. At times it may be detected only on the points of gum between the teeth. When present in one jaw alone, it is more apt to be seen in the upper than the lower. Garrod and Oliver have described a bluish



discoloration of the mucous membrane of the lips where it has been in contact with an accumulation of tartar on the teeth.

The colicky pains of lead poisoning occur in paroxysms. Generally, the pain is centralized about the umbilicus, although it may be either diffused, or present several foci of greatest intensity. While the paroxysmal pain continues, the abdominal walls are retracted. Pressure generally relieves this pain. A very remarkable accompaniment of lead colic is the change in the character of the pulse. It becomes greatly reduced in frequency, and heightened in tension. Between the paroxysms of colic the patient suffers from a dull, heavy abdominal pain, aggravated by pressure. The bowels are obstinately constipated. The nature of lead colic has not been definitely determined. There is every reason, however, to believe that it is due to a tetanic contraction of the colon, dependent upon the action of lead on the sympathetic ganglia and the muscular coating of the bowel. Oliver has called attention to an inequality of the pupils during the course of lead colic.

The deleterious influence of lead on the sexual system is not as well known as it should be. Among women, ovaries and uterus are alike affected. Menstrual derangements, amenorrhœa or menorrhagia are almost invariably present. The pregnant exhibit a remarkable tendency to spontaneous abortion. Even when the patient goes to full term, the child is apt to be sickly, and dies in early infancy. The evil effects of lead on the fœtus are exhibited even in cases in which the father only is the sufferer from it. It has been shown that even the lower animals chronically poisoned by lead suffer from deranged sexual functions.

Neuralgic pains are common phenomena of lead poisoning, but present no especially characteristic features. They are usually described by the patient as "rheumatic," and are situated apparently in the muscles or the joints. Sometimes the pains assume a distinctly neuralgic character, and invade the limbs. Cramps in the calves of the legs are not unusual.

Lead paralyses exist in quite a variety. The most characteristic and the form most frequently encountered is a double wrist drop—double because affecting both sides alike. It is due to paralysis of the muscles to which the musculo-spiral nerves are distributed, the long supinators alone escaping. Its onset is somewhat rapid, there being required a period ranging from a few days to three or four weeks for its full development. It usually makes its appearance on one side first, the right in right-handed persons, and the left in the left-handed. The power of extending the wrist is lost, excepting when the fingers are held flexed. In the latter position, the proper extensors of the wrist are capable of acting, these muscles not being affected so early or so completely as the other extensors. Atrophy of the affected muscles soon follows the onset

of the paralysis. This is evidenced by the flattening of the back of the forearm. Electrical examination shows the reaction of degeneration to be present. It may even be that the qualitative changes in the galvanic reaction may precede the paralysis by some little time. The increased galvanic irritability of the paralyzed muscles usually passes away within a few months. In aggravated cases going on to more or less complete atrophy, galvanic as well as faradic irritability may be ultimately destroyed.

The form of lead palsy, above described, is typical of the first of the two types of lead paralysis described by Gowers. In a general way, the class of which this is a representative, is characterized by localized paralysis, the paralysis preceding the muscular atrophy, and being associated with the reaction of degeneration. The second variety closely resembles progressive muscular atrophy, the atrophy appearing synchronously with the loss of power, and faradic and galvanic irritability of the muscles proceeds *pari passu* with the muscular wasting. The paralysis and wasting almost invariably begin in the small muscles of the hands. Returning to our description of the first class, we do not always find the paralysis with the distribution just described. It may invade the muscles of the upper arm. In this case, the deltoid, biceps, brachialis anticus, supinator longus, and sometimes also the supra- and the infra-spinatus. The deltoid muscle is usually the first muscle to suffer in these cases. Buzzard has described a case, in which it was the only one affected. In still other cases, the muscles supplied by the perineal nerves are affected, the tibiales antici alone escaping.

Gowers's second variety of lead palsy is generally associated with fibrillary twitchings in the affected muscles. It is very obstinate to treatment, and often persists long after exposure to the poison has ceased.

Oliver has described a form of lead paralysis, in which the muscles of an extremity are affected *en bloc*. It is of rapid onset, large areas of the body being rendered helpless within a day. Even the muscles of the trunk, abdomen, and thorax become powerless. The muscles of the head and neck escape. As a rule, these cases make a rapid recovery. Some of them, however, end fatally.

Tremor is not a very frequent symptom of lead poisoning. When present, it usually affects the hands. It is nearly always fine in range, and is aggravated during voluntary movements.

The influence of lead on the brain is quite varied. In many cases, the action on this organ is manifested by symptoms apparently of hysteroid nature. Hemianæsthesia, hemiplegia, hemianopsia, amaurosis, hysteroid seizures, and general neurasthenic symptoms are the principal source of complaint. Sometimes, mental symptoms of an organic origin are present. These include true epileptic convulsions, insomnia, general restlessness, delirium, coma, and even insanity. The latter,

though rare, generally assumes the type of melancholia with delusions. Progressive mental failure has, however, been observed, and Manakow and Ullrich have reported cases closely simulating general paralysis of the insane.

Optic neuritis and atrophy of the optic nerve are rare symptoms of lead poisoning. An acute neuro-retinitis sometimes accompanies lead encephalopathy. Sometimes, an optic neuritis occurs in conjunction with headache and vomiting. Sight is rapidly lost, but may be regained. In other cases, the neuro-retinitis is a secondary symptom, accompanying, for example, kidney disease. It then presents all the appearances of retinitis albuminurica. Occasionally, an apparently idiopathic atrophy of the optic nerve results from lead poisoning. It goes on to complete loss of sight.

Of the effects of lead on the cranial nerves, those relating to the optic nerve have just been mentioned. There may be mentioned, in addition, palsy of the ocular nerves. Mackenzie has reported laryngeal palsy from lead.

Lastly, the effect of lead on the kidneys is to be considered. These organs at first take on a parenchymatous inflammation, later becoming interstitial. The deleterious action of lead on the kidneys is probably not sufficiently realized, because the pathological changes set up by it continue to progress after all other evidences of the intoxication have ceased. It may even happen that the kidneys give not the slightest evidence of unsoundness during the early stages of chronic saturnism, and the manifestations of interstitial nephritis only appear years subsequently.

The relation of lead poisoning to gout has always been an interesting subject of study in England. Lead poison undoubtedly awakens gouty symptoms in those predisposed, probably because of the deficient elimination of uric acid thus caused.

**Diagnosis.**—The recognition of lead poisoning rests upon a knowledge of the possibility or probability of the ingestion of the poison, together with a recognition of symptoms known to arise from this cause. The blue line along the gums, the colic and the paralysis, are the most important of the latter. If examination of the urine discloses the fact that lead is being eliminated by the kidneys, then the diagnosis becomes conclusive. It is customary with text-books and teachers to state that lead can be recognized by the usual tests—very unsatisfactory information truly, for lead can only be detected in the urine after a most painstaking investigation. Taylor gives the following directions for this purpose: The urine should be evaporated to dryness and “incinerated in a porcelain vessel. The ash should be heated with a small quantity of nitric acid, and then evaporated to dryness. The nitrate of lead thus formed may be dissolved out of the residue by water and filtered. A portion of liquid evaporated on a slide will yield crystals of nitrate



of lead, which may be identified: (1) By covering them with a solution of iodide of potassium. If lead, they acquire a brilliant yellow color. (2) A solution of sulphide of ammonium renders them black. The remainder of the liquid, after filtration, may be treated with a current of sulphuretted hydrogen gas. A brown color, or a brown precipitate, not readily dissolved by nitric acid, indicates the presence of lead." The method proposed by Prof. Wood, of Harvard, is even more delicate; but is open to the objection of being unduly complicated for clinical purposes. It is as follows: The patient takes five or ten grains of iodide of potassium three times daily for four or five days, and then collects a quart of urine, "which is acidified with acetic acid, and sent for examination. The analyst first evaporates the urine to dryness, and fuses it in a crucible with a little pure nitre till it becomes white. When the crucible is cool, dilute hydrochloric acid is added hot, to extract the residue. It is then filtered, and the filtrate treated with ammonia to alkaline reaction, to precipitate the phosphates and iron. Ammonium sulphide is added at the same time, to throw down the lead and iron as sulphides. This is washed three times by decantation with hot water; then water acidified with hydrochloric acid is added, and the whole allowed to stand until the next day. It is then filtered through a small filter, the filtrate rejected and the residue washed. A little pure nitric acid is then added, drop by drop, to dissolve the lead left on the filter and to carry it through as nitrate. The filtrate is then collected in a watch-glass and evaporated to dryness, and the final test made by adding a drop of water and a crystal of potassium iodide. The production of a yellow precipitate denotes lead." The recognition of lead in the urine requires a short course of iodide of potassium, in order that the poison may be eliminated by the kidneys.

**Prognosis.**—As a rule, this is favorable. It is necessary, however, that the exposure to the poison be stopped. Second attacks are far more easily brought on by renewal of the exposure. Death is of occasional occurrence.

**Treatment.**—The successful treatment of a case of lead poisoning necessitates a recognition of the cause of the trouble. Exposure to lead must be stopped. Iodide of potassium in small doses, administered for the purpose of eliminating the lead by the urine, must be given. Three grains of the drug three times daily should be sufficient. General and local massage is a valuable adjuvant. Accurate clinical experimentation has demonstrated that massage is the most efficient means for the elimination of lead at our disposal. Other treatment should be symptomatic. Lead palsy finds its most efficient treatment in galvanization of the paralyzed muscles. *Opium* is usually the most efficient remedy for the constipation and the colic. *Alumina*, *belladonna*, *nux vomica* and *platina* are other remedies sometimes useful for the colic. These remedies are likewise of value for the other symptoms.

# AFFECTIONS OF MUSCLES.

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## MYOSITIS.

**Nomenclature.**—Myositis unquestionably means an inflammation of muscle; and yet the term is one capable of giving rise to considerable confusion because of the variety of diseases designated by this name, all of them muscular inflammations. Thus we have a simple myositis arising usually from traumatism, or occurring secondarily to infective processes, or as an associated condition in certain of the infectious fevers, as erysipelas, typhoid fever, glanders, and diphtheria. Again there is a variety of muscular inflammation of almost universal distribution, of probably rare occurrence, and probably dependent upon infection by a specific microbe, the nature of which is as yet unknown; these cases should be designated *acute polymyositis*. *Myositis ossificans progressiva* is a variety of muscular disease in which the affected muscles undergo calcification.

**Symptomatology.**—The characteristic symptom of myositis is pain, swelling, and œdema, affecting the muscular structures. In acute polymyositis this is so universal as to suggest very strong resemblance to the muscular pains of trichinosis, with which condition it is not separable without a microscopic examination of fragments of the affected muscles. In acute polymyositis the extremities are swollen and painful; later the pathological process extends to the muscles of respiration and deglutition. In the majority of cases, the disease is associated with an erythematous eruption affecting the trunk and extremities. The spleen is usually swollen. In the later stages of the disease, bronchitis and pneumonia are liable to develop and add greatly to the patient's distress because of the exertion and pain attending expectoration.

**Prognosis.**—Acute simple myositis offers a favorable prognosis in the majority of cases. Much depends upon the etiological influences involved in the case. Often suppuration ensues, and must be treated surgically. Acute polymyositis is a very serious disease, nearly all of the cases dying in the course of three or four weeks.

**Pathology and Morbid Anatomy.**—Examination of the muscles shows them to be the seat of all varieties of degeneration and destruction. Foci of inflammation are found in the interstitial connective tissue. The nerves themselves are perfectly normal.

**Treatment.**—The treatment of myositis of all varieties must be conducted on the general principles involved in the management of inflammatory affections generally. In the early stages, *aconite*, *gelsemium*, *bryonia*, and *belladonna* will be found indicated, according to the special characteristics of the inflammation. In suppurative cases, *hepar*, *silicea*, and *mercurius* are suitable. In polymyositis, *bryonia*, *belladonna*, *arsenicum*, *lachesis*, *phosphorus*, and *aurum*.



## THE MUSCULAR ATROPHIES.

The consideration of the various diseases included in the title, "Muscular Atrophies," is a subject which I approach with much diffidence. A careful study of many authorities discloses remarkable variations in opinion. Classification after classification has been proposed, sometimes with, but oftener without, reason, thus serving to greatly confuse the minds of even the most expert. Of certain facts we are assured. In the first place, it is certain that some of the types of progressive muscular atrophy are dependent upon spinal and some upon primary muscular lesions. The types of disease represented in each of these two classes present the widest differences one from the other. When, however, one comes to study the different conditions making up each of these classes, confusion prevails. Considering those of spinal origin, one finds described a chronic poliomyelitis anterior, a progressive muscular atrophy, a bulbar or glosso-labial palsy, an amyotrophic lateral sclerosis and a cervical hypertrophic meningitis. Under the head of the primary muscular atrophies, now generally called muscular dystrophies, one finds a number of disease types presenting certain clinical features, but the pathological nature of which may thus far be regarded as largely hypothetical.

It is the tendency of some authors to regard chronic poliomyelitis as the essential lesion, in fact, the only primary lesion, in the case of the various diseases above enumerated, as including the spinal muscular atrophies. However strongly the microscope may assign to these various conditions the same morbid changes distributed in various portions of the central nervous system, the clinical pictures presented by them, together with the different courses they pursue, seem to indicate that they must have different pathological foundations. For example, it seems to me that there is a strong distinction between chronic poliomyelitis and progressive muscular atrophy (spinal type). Otherwise how can we explain certain characteristic differences in their symptomatologies; and how account for the frequency with which one recovers, and the other progresses to a fatal termination? In both instances the lesion is limited to the ganglion-cells of the anterior cornua. At the time of the investigation the microscopic appearances are practically identical. This does not teach, however, that the appearances have been the same throughout the various stages of the two diseases.

As to the muscular dystrophies, still wider variations have been announced. Erb has described his type of juvenile muscular atrophy;

Landouzy and Dejerine have described a type, also known as the scapulo-humeral type, and to which their names have been given. Leyden has endeavored to construct a hereditary type, which, in view of the fact that nearly all the clinical conditions comprised in the other types are hereditary, seems altogether superfluous. The Landouzy-Dejerine type may, likewise, be regarded as an excess of clinical refinement, for a study of the cases shows that they present but the ordinary features of Erb's juvenile form, with the addition of preliminary wasting of the facial muscles. As to the identity of pseudo-hypertrophic paralysis and Erb's type, considerable discussion has been had. Certainly, the two present many features in common, and yet withal, certain wide differences. When muscular atrophy complicates the former, it attacks the same muscles which it involves primarily in the latter.

Study furthermore teaches us that still another type, the peroneal form of muscular dystrophy, described by Charcot and Marie, and independently of them by Herbert Tooth, is variously claimed as of spinal, peripheral, and muscular origin. The present trend of opinion favors the view that it is either a poliomyelitis anterior chronica lumbalis (Sachs), or a peripheral neuritis (Gowers).

Bearing in mind all this confusion respecting the morbid anatomy and pathology of the muscular atrophies, I feel that the interests of my readers will best be served if I neglect all classifications founded upon such uncertainties, and base my description exclusively upon clinical types.

Regarding the features of the muscular dystrophies, I would say that they are essentially family diseases. They seem to be dependent upon some congenital defect. They are rarely observed in this country, with the exception of pseudo-hypertrophic paralysis.

### CHRONIC POLIOMYELITIS ANTERIOR.

**Etiology.**—Most cases of chronic poliomyelitis anterior occur between the ages of thirty and fifty. The causes, so far as we know them, appear to be exposure to damp and cold, excesses in alcohol and venery, lead poisoning and syphilis.

**Symptoms.**—The paralysis always makes its appearance first in the lower extremities, showing itself as a weakness or a tired feeling on standing, this gradually increasing until a distinct muscular weakness is apparent. These early symptoms are often accompanied by fever, gastric disturbances, etc. Slowly the muscular weakness, sometimes involving one leg, sometimes both, increases until finally there is complete paralysis of individual muscles or sets of muscles, or even of one or both lower extremities entirely. Following closely on the paralysis is an atrophy of the affected muscles. Fibrillary twitchings are observed in the early stages. Then the disease extends to the upper extremities. In so doing

it shows but little preference for attacking one set of muscles more than another, though the muscles of the fingers and hands are apt to be more severely affected than are the large muscles of the arm and shoulder. The sensory functions and the sphincters are never involved. Reaction of degeneration is always present. The deep and superficial reflexes are early impaired or destroyed.

**Diagnosis.**—There is danger of confusing chronic poliomyelitis anterior with progressive muscular atrophy. The latter affection is readily differentiated in that the wasting always begins in the upper extremities, and is the direct cause of the loss of power; there is no reaction of degeneration; and the deep and superficial reflexes are only diminished when the muscular atrophy is profound. Chronic poliomyelitis runs a more rapid course, and often ends in recovery; while progressive muscular atrophy is of much slower onset, and pursues a steadily downward path. It is likewise to be differentiated from multiple neuritis, which is attended by anæsthesia and by pain and tenderness along the course of nerve trunks.

**Prognosis.**—A large percentage of cases of chronic poliomyelitis anterior recover. The muscles last affected are usually the first to regain their functions. In fatal cases the disease runs a course of from one to four years. In quite a number of the cases, some of the muscles remain permanently paralyzed.

**Treatment.**—Galvanization of the spinal cord and the administration of such remedies as those recommended in the treatment of acute poliomyelitis and progressive muscular atrophy will give the best results to be obtained.

### PROGRESSIVE MUSCULAR ATROPHY.

Progressive muscular atrophy is a disease having its primary pathological changes in the spinal cord, and characterized by wasting of individual muscles, or groups of muscles, slowly progressive both as to degree and extent of the atrophy, and unattended by any sensory phenomena of importance.

**Etiology.**—Males are more frequently affected than females, probably because they are more subject to the exciting causes of the disease. It has been noted that women whose occupations force them to withstand exposure and fatigue, are no more exempt than males. The spinal form of progressive muscular atrophy is almost exclusively a disease of adult life, attacking patients between the ages of twenty-five and forty-five years. The influence of hereditary causes in predisposing to the disease is a debatable question. Neuropathic predisposition is undoubtedly a powerful factor in very many instances. Cases exhibiting a remarkable direct heredity are almost certainly to be classed as essential muscular atrophy.



Among the exciting causes, mental distress, exposure to cold, and over-exertion are to be noted. In the case of exposure and over-exertion it is an interesting fact that the atrophic process begins in the part which has been most directly exposed to the exciting influence. Injury has caused the disease in some rare instances. A concussion of the spine has been followed by atrophy of certain muscles, and this atrophy slowly spreads. In other cases a blow on a single muscle starts the disease. Syphilis and lead poisoning must also be borne in mind as occasional causes.

**Symptoms.**—In some instances, the disease is ushered in by dull rheumatoid pains, sometimes situated in the parts about to be wasted, sometimes limited to the spine. Especially is this method of onset observed in cases arising from exposure to cold. In the majority of cases, the disease begins very insidiously. The patient notices that certain movements are not performed with accustomed ease, and directing his attention to the parts, observes local wasting. In nearly all cases, this wasting begins first in the upper extremities, the muscles of the thenar and hypothenar eminences being the first affected. The interosseous muscles suffer, thus causing depressions between the metacarpal bones, and bring the flexor tendons into prominence. Next, the disease spreads to the forearms, involving the flexor more than the extensor muscles. This peculiar method of onset causes the hand to assume the deformity known as the claw-shaped hand, or the "*main en griffe*."

In some instances, the atrophy is first noticed about the shoulder, the deltoid being the first muscle to suffer. In still others, the forearm affords the first evidences of the disorder. In rare cases, the legs first atrophy, and then the wasting proceeds in them to a more marked degree than in any other part of the body. This is the reverse of the condition observed in cases beginning in the upper extremities, in which the muscular wasting in the legs does not usually proceed to a very high degree, though the loss of power in them may be great.

Certain muscles, different portions of which have different functions, do not waste in their entirety, as, for instance, the trapezius and the pectoralis major. The muscles of respiration rarely escape. The intercostals, the accessory respiratory muscles, and the diaphragm, one or all, may be involved.

The tendency of the atrophy is to increase gradually until all muscular fibre is destroyed. Even the subcutaneous fat may disappear, leaving the affected part to consist literally of only skin and bone.

Peculiar subcutaneous twitchings, known as fibrillary twitchings, are very frequent accompaniments of the atrophic process. When not present, they may frequently be excited by a slight tap on the muscle, or by exposure of the part to cold air.

The deep and superficial reflexes are altered according to the severity

of the atrophic process. The same is true of the electrical reactions. As long as a healthy, muscular fibre exists, just that long is some electrical irritability of both nerve and muscle retained.

Sensory and visceral functions, and the control over the sphincters are retained.

The symptomatology of progressive muscular atrophy may be varied from the typical, as above outlined, by spread of the pathological process to the lateral columns, and when this occurs, we have added certain rigidities of various degrees of intensity and distribution.

**Diagnosis.**—From *chronic poliomyelitis*, progressive muscular atrophy is to be distinguished by the data presented when describing the former affection.

*A limited muscular atrophy*, due to a neuritis, generally the result of traumatism, may be confounded with progressive muscular atrophy. But in such cases the atrophic process is confined to muscles supplied by single nerve trunks, and it is associated with impairment of sensation. The onset is likewise more rapid than in the case of progressive muscular atrophy.

*Lead poisoning* may be recognized by the history of exposure of the patient to that influence, the blue line on the gums, the previous history of lead colic, the distribution of the paralysis, and the comparatively rapid mode of onset.

*Multiple neuritis* is attended by paralysis with muscular atrophy; but in such cases the reaction of degeneration will be found; disorders of sensation in the shape of pain along the course of the nerve trunks and impairment of sensation are necessary accompaniments.

**Prognosis.**—The outlook in a case of progressive muscular atrophy is necessarily bad. Yet quite a number of cases cease to progress some time during the course of the disease. Unfortunately, however, this result is only attained spontaneously when the muscular atrophy has reached such a high degree as to reduce the patient to almost complete helplessness. Careful treatment may bring the disease to a standstill at almost any time. Cases in which the atrophy spreads symmetrically are said to offer a better prognosis than do others.

**Treatment.**—Mild methodical exercise, fresh air, and good food are essential. Electricity is an important agent also. It is to be employed centrally to the spine, and locally to the atrophying muscles. The galvanic current may be applied directly to the spine, the positive electrode over the cervical enlargement, and the negative to the lumbar region; or the plan suggested by Erb may be pursued. He advises the application of galvanism to both the spinal cord and the sympathetic. "His method is to commence with the anode to the cervical spine and the cathode to the cervical sympathetic, followed by the cathode to the spine, and the anode to the sternum, the lumbar enlargement, or the

peripheral nerves. He insists especially upon the importance of the action of both poles being brought to bear successively upon the affected regions of the cord. Finally the affected muscles are to be galvanized or faradized, the indifferent electrode being at the nape of the neck. The current should be moderately strong, but too vigorous treatment is not advisable." As to peripheral applications, the best results are to be obtained by the interrupted galvanic current. The strength should be just such as to produce muscular contractions. Duchenne is a firm believer in the efficiency of faradism. His instructions are as follows: "(1) To pass the moistened electrodes over the surface of each of the affected muscles, keeping them close together, and using a current of low intensity (primary current). (2) To stimulate the muscles moderately, and with a current which is not interrupted very frequently. (3) To treat only the muscles which react to faradism, and to pay most attention to the most important muscles, and to terminate the sitting by a mild faradization of any muscles which may be threatened with an invasion of disease."

Gowers praises very highly the hypodermic administration of strychnia, claiming that the course of the disease is generally stopped within one month after the commencement of the treatment.

The nitrate of strychnia is the most convenient preparation. One hypodermic administration is made daily. The initial dose is one one-hundredth of a grain rapidly increased until finally one fortieth of a grain is given.

*Plumbum* is perfectly homœopathic to progressive muscular atrophy, and has achieved favorable results in a number of cases. *Argentum nitricum* likewise is capable of producing good results by reason of its beneficial influence over spinal degenerations generally.

*Phosphorus*, *arsenic*, *arnica*, *mercurius*, *gelsemium*, *physostigma*, *sulphur*, *baryta*, and *cuprum* may also be studied in this connection.

## IDIOPATHIC MUSCULAR DYSTROPHIES.

(A) **ERB'S JUVENILE MUSCULAR ATROPHY.**—The term "juvenile muscular atrophy" as applied to this affection is somewhat of a misnomer, as the disease not infrequently attacks patients pretty well advanced in years, although the majority of cases begin in childhood and youth. The muscles about the shoulder are the first affected. Later those of the upper arm become involved. The muscles especially apt to be affected are the pectorals, trapezii, rhomboids, serrati, latissimus, longissimus. The muscles of the forearms, the deltoid, the supra- and infra-spinati usually escape. When finally the atrophy involves the lower extremities, the muscles about the hips and thighs are first affected. The legs and likewise the forearms do not suffer until quite late, or escape entirely. The diaphragm may atrophy and thus be the cause of



death. The peculiarities of gait and posture of pseudo-hypertrophic paralysis are frequently present in this disease. Of the significance attached to this observation I have already spoken when treating of the clinical aspects of the muscular atrophies in general.

There are no fibrillary contractions of the affected muscles. The electrical reactions are only altered in the direction of intensity of the response, which is directly proportional to the volume of the degree of wasting in the several muscles affected.

The course of the disease extends over many years. Erb has described one case which lasted thirty-eight years.

Erb's type of muscular atrophy is rarely seen in this country.

(B) LANDOUZY-DEJERINE TYPE OF MUSCULAR ATROPHY.—This is also spoken of as the facio-scapulo-humeral type of muscular atrophy. Like the preceding, it is dependent upon hereditary influences very largely. It usually begins in childhood, at an earlier age than Erb's muscular atrophy, usually about the third or fourth year. It may, however, develop later in life. The atrophic process begins first in the face. The wasting of the orbicularis oris causes the lips to protrude, giving rise to the facial deformity which the discoverers of the disease described as "the tapir-mouth." The muscles of the eye, deglutition and mastication escape. The atrophy then extends to the muscles of the shoulders and arms, and pursues the course of Erb's type of muscular atrophy.

(C) PERONEAL TYPE OF CHARCOT AND MARIE, AND TOOTH.—In this, the atrophy begins first in the extensor longus pollicis, extensor digitorum communis, or the peronei muscles. It then gradually extends. Sometimes the intrinsic muscles of the foot are affected; in still others, the gastrocnemius. The unequal wasting of the muscles gives rise to various forms of club-foot. The disease spreads symmetrically. After many years it involves the upper extremities, attacking first the intrinsic muscles of the hands.

Fibrillary contractions of the atrophying muscles are noted, and reaction of degeneration in a modified form is sometimes present. There is no doubt concerning the occasional impairment of sensibility, and the presence of pain. The knee-jerks are preserved when there is considerable atrophy below the knees, providing the quadriceps extensor is intact. The cutaneous reflexes present considerable variations. They are preserved, however, much oftener than one would expect *a priori*.

**Diagnosis.**—The main difficulty in the recognition of the primary muscular dystrophies, is found in their differentiation from the *spinal type of muscular atrophy* and certain forms of *multiple neuritis*. From the former we are enabled to distinguish them: (1) By the history of heredity, which is altogether absent in the spinal progressive muscular atrophy. (2) By the age; the primary dystrophies coming on early in life, progressive muscular atrophy occurring mostly in adults. (3) By the

peculiarities in the distribution of the wasting. (4) By the presence or absence of fibrillary contractions, and the condition of the electrical reactions. (5) In many cases of the spinal form, there is a spastic condition of the lower extremities with increased knee-jerks, etc.

In atrophy from multiple neuritis, reaction of degeneration, sensory disturbance, and pain are marked features. The paralysis precedes the atrophy.

**Prognosis.**—The prognosis in primary muscular dystrophies is positively bad. They run a very chronic course, lasting usually for many years. No case, so far as is known, has ever been cured.

**Treatment.**—In outlining a course of treatment, one must be guided largely by theoretical considerations. Prophylaxis is the essential feature. The markedly hereditary character of the ailment makes it incumbent upon members of affected families to refrain from marriage. In a general way, most careful attention should be paid to all the details that make up a hygienic course of living. When once the disease has appeared, treatment should, in the present knowledge of the subject, be conducted on purely general principles; mild massage and gymnastics, and the administration of such remedies as seem to be called for from time to time by existing conditions.

### PSEUDO-HYPERTROPHIC PARALYSIS.

This is a variety of myopathic muscular atrophy in which there is a false hypertrophy of certain of the affected muscles, unassociated with sensory, sphincteric, or mental disturbances.

**Etiology.**—All that is known respecting the etiology of pseudo-hypertrophic paralysis is that it shows a most remarkable tendency to attack the children in certain families. This hereditary tendency may run through several generations. Boys, in particular, are affected. The disease is usually transmitted by such of the female members of the family as become mothers. The first signs of the trouble are generally noticed at about the age of four or five years, rarely after the age of ten, and never after twenty.

**Symptoms.**—In some cases, the existence of pseudo-hypertrophic paralysis is first recognized by the failure of the child to walk properly when it arrives at the proper age. In others, attention is first directed to certain acquired peculiarities in gait. In all, the apparently well-developed muscles are not looked upon as pathological until other symptoms assert themselves. The disease is invariably of very slow onset and progress. At first the only abnormality noticed is a tendency to fall from trifling causes. Especially does the child exhibit a difficulty in ascending stairs, taking hold of the banisters to pull himself up. Then a peculiar waddling gait is noticed. In walking, the pelvis is raised by the bending of the body towards the side of the passive limb, a step is

taken, and then the body is swayed to the opposite side. When standing, the patient is noticed to tilt the pelvis markedly forward. This makes the abdomen prominent, and the lumbar spine decidedly concave, a condition known as lordosis. The upper portion of the body is thrown backwards, so that a plumb line let fall from between the scapulæ clears the sacrum by two or three inches. This lordosis disappears entirely when the patient is seated. When the child attempts to rise after sitting on the floor, he does so in a very characteristic fashion. First he gets on both hands and knees; he then raises himself by straightening out all four extremities, thus resting on hands and feet, with his body arched. He then gradually brings his different points of support closer and closer together. Next one hand and then the other grasp the corresponding leg, thus supporting the body. Then he begins the process called "climbing up the thighs." By placing the hands higher and higher, first on the legs, and then on the thighs he is enabled to bring himself to an erect posture. Certain muscles, notably those of the calves, are found to present an unnatural enlargement. Sometimes, this false hypertrophy is widespread, and gives the paralyzed child an appearance of herculean strength. The sphincters are never involved. Sensory disturbances are absent. The knee-jerks are normal in the early stages as are also the electrical reactions.

**Pathology.**—This disease is a primary affection of the muscles themselves, the alterations occasionally found in the spinal cord being secondary and occurring only after a number of years. There is an overgrowth of connective tissue, and a deposit of fat cells in the muscular substance, with subsequent atrophy of the muscle fibres themselves. It has been pretty well established that the foundation of the disease is laid at birth.

**Diagnosis.**—The peculiarities in gait and the false muscular hypertrophy are simulated by no other condition. By many authorities the former are looked upon as decidedly more characteristic than the latter.

**Prognosis.**—This is most emphatically unfavorable. The course of the malady is remarkably slow, gradually increasing to complete helplessness, but often lasting for many years, sometimes as long as forty. The sufferer usually dies of some intercurrent illness, generally some disease of the respiratory apparatus.

**Treatment.**—Little if anything can be expected from electricity; indeed, some authorities go to the extent of pronouncing this latter agent injurious in pseudo-hypertrophic paralysis. Massage, on the other hand, is praised by all. It must not be pursued too energetically, however. The manipulation should be just sufficient to excite a beneficial hyperæmia of the weakened muscles. Supplementary to massage is systematic but gentle exercise, which must tend to preserve muscular nutrition, and at least lessen the rapidity of progress of the pathological process.



Owing to their well-known influence over fatty degenerations and connective tissue overgrowths, *iodide of potassium* and the preparations of *gold* are suggested as the most available remedies. Their dosage need not be limited to the minute or infinitesimal, however. The former drug may be given in doses of from fifteen to thirty grains daily, and kept up for months or years. The latter may be administered either as a *muriate*, or as the *double chloride of gold and sodium*. *Phosphorus* should likewise be a valuable remedy. Indeed, Dr. J. Galley Blackley reports one case in which this remedy did very good work. The same author also reports a case in which lathyrus effected an improvement which was but temporary.

## THOMSEN'S DISEASE.

**Synonym.**—Myotonia congenita.

**Definition.**—A disease characterized clinically by tonic spasms of voluntary muscles, taking place only on attempted willed movements. Its pathology is uncertain.

**Etiology.**—Thomsen's disease is especially characterized by its tendency to run in families. The name by which it is most commonly known was given it because the first complete account of the disorder was published by a Dr. Thomsen, in whose family it had existed for several generations. The total number of cases reported is not large, most of these occurring in Germany and the Scandinavian Peninsula. But three cases have been reported in the United States. Most of the cases occur in males. The symptoms generally make their appearance in childhood.

**Symptomatology.**—The onset of the disease is gradual, the difficulty in executing voluntary movements being as a rule attributed to awkwardness. As soon as the patient attempts to make a movement the muscles concerned become rigid, and the limb is held motionless. After several seconds the muscles relax and the movement is accomplished, the rigidity reappearing on the attempted execution of the next movement. In some cases the rigidity is limited to the muscles concerned in voluntary action; in others it becomes universal after the long continuance of the disease. It sometimes happens that difficulty is experienced only when beginning the movements; that is to say, the patient finds it difficult to walk immediately on rising from a sitting or lying posture, but meets with little trouble after he once gets started. The difficulty is mostly limited to the muscles of the extremities, although in rare instances, those of the face and eyeballs become involved. The tonic contractions are always painless. Sensory symptoms are absent. The deep and superficial reflexes are normal. Mental deterioration sometimes takes place. The affected muscles give the appearance of good development and great strength; but in reality they are weak. The tonic contractions are aggravated by cold and mental excitement. They are much relieved by judicious exercise.

**Prognosis.**—Thomsen's disease is incurable. Some cases are stayed in their progressive course.

**Treatment.**—There is no treatment known to have any influence on the course of the disease, other than the advantage of regular and systematic exercise.

## APPENDIX.

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Since the printing of the article upon diphtheria, the very valuable communications upon this subject, presented to the Eighth International Congress of Hygiene and Demography at Buda-Pesth, have appeared, containing valuable information relating especially to the etiology and treatment of this affection. The following summary of the paper of Dr. F. Loeffler will be found to be a concise statement of our knowledge of the causes and prophylaxis of diphtheria :

“(1) The productive agent of diphtheria is the diphtheria bacillus. Dispute as to the etiological definition of this bacillus exists no longer. We can, therefore, henceforth indicate as diphtheria such forms of disease as are infested with the bacillus.

“(2) Not infrequently cases appear in the early stages to the clinical observer as true diphtheria, which, however, are caused by other organisms, as streptococci, staphylococci, pneumococci, and in light or graver form may be mistaken for diphtheria. But the differential diagnosis can be effected through bacteriological research. Statistical complications on the epidemic spread of diphtheria, as well as on the character of diphtheritic epidemics, cannot represent an exact definition so long as the bacteriological investigation of cases suspected of diphtheria fails to mark a division between true diphtheria bacillus and cases merely resembling diphtheria.

“(3) Diphtheria epidemics show a various character, as do many other epidemics of infectious disease. The course of the epidemics is often very light, but also much more severe, indicated by the high figure of the death-rate, the rapid infection of the larynx and the nose, and by severe heart and kidney affections, and consecutive paralyses. But also in the same epidemic instances of severe and light forms of disease frequently alternate irregularly.

“(4) The variation, of course, will be determined by several factors: (a) By differences in the number and the virulence of the diphtheria bacilli; the causes of the latter are not yet absolutely known. (b) By concomitant bacteria, and, indeed, as much by pathogenic as saprophytic; the processes of infection with regard to the diseased mucous membranes in the passages and in the nose appear to influence the course of the disease unfavorably, in part by increasing the virulence of the bacilli,



in part by weakening the body through absorption of decomposition products. (c) By individual tendencies not yet thoroughly recognized.

“(5) The diphtheria bacillus can appear in the passages, especially of the nose, of separate individuals without causing indications of sickness, which it first induces when it has actually established itself. Lesions of the mucous membranes, small eruptions, catarrhal changes, are favorable to its residence. In brief, meteorological conditions, giving admission by the first approach to catarrh, especially cold, damp weather, appear to favor the sickening from this cause. But this influence has to be more closely observed.

“(6) Diphtheria is most rapidly communicated by direct contact between sick and well through spitting, coughing, sneezing, kissing, and grasping of the hands, whereby the hands come into contact with fresh secretion, but also freely through utensils which the sufferer has fouled with his excretions, by beverages, food, eating and drinking vessels, cast-off washing clothes and other articles, as pocket-handkerchiefs, playthings, even long after their actual infection.

“(7) The sick is infectious so long as he has bacilli upon the mucous membranes. The bacilli usually disappear with or soon after the disappearance of the local signs, but they may be detected still lively and virulent in the passages or nose for weeks and even months.

“(8) In organic matters condensed and excluded from light the bacilli can maintain themselves for a period of months outside the body; accumulations of dirt, dark and close dwellings favor thus the preservation of bacilli and the extension of disease.

“(9) As a specially noticeable vehicle for the extension of disease is to be noted the crowding together of susceptible individuals, especially in families of many children. But other gatherings of people, apart from children, where separate persons do not come into such proximity as the members of a family, may offer facility for the extension of infection, as schools, barracks, and the like.

“(10) The diphtheria bacillus is so far not identified with certainty as the cause or inducer of other diseases similar to diphtheria or of other spontaneous disease of lower animals. The possibility of the conveyance of true diphtheria from sick animals to human beings is thus outside our present knowledge. It is desirable that the Governmental investigating committees should combine with research regarding diphtheria coming under their notice the similar diseases of animals, and also the communication from animals to human beings of diseases resembling diphtheria.

“(11) As prophylactic means are to be considered : (a) Care for cleansing, keeping dry, sufficient ventilation, and lighting of the dwelling; (b) careful cleansing of the mouth and nose, gargling with weak solutions of common salt and carbonate of soda, thorough brushing of the teeth,

extraction of bad teeth, attention to the deeper cavities of the tonsils, and removal of hypertrophied tonsils; (c) cold douching of the throat in times of diphtheria prevalence.

"(12) Every case suspected as diphtheria must, when possible, be bacteriologically investigated. The physician must have easy access to the required materials for carrying on the culture, for example, in the chemists' shops. The investigation has to be carried on by specialists, as in the case or cases of suspected cholera.

"(13) All cases proved bacteriologically to be true diphtheria, as well as all cases suspected as diphtheria which have not been bacteriologically investigated, must be dealt with as under police regulations.

"(14) Every diphtheria case must be isolated either in a separate room of the dwelling or in an isolation ward. In order to restrict as much as possible the spread of the bacilli by the sick, a local anti-bacillar treatment must be employed with a view to prophylaxis against the early stages of the disease.

"(15) One of the most effective means against the spread of diphtheria to be cared for is the protective inoculation of susceptible individuals in the neighborhood of the patient, especially of children. In proportion as the innocuousness of Behring's serum cure through preventive injection is established for curing or prophylaxis, it appears worth while to develop further as far as possible the art of inoculating it in families and in school classes in which diphtheria cases have occurred.

"(16) In every case of diphtheria disinfection is imperative. This is needed for all utensils for the sick, as well as for the sick themselves and the sick-room.

"(17) Convalescents from diphtheria must not mix freely with others (or children go to school) till bacteriological investigation has proved the removal of the bacilli, and the sick after a warm bath with soap have been thoroughly cleansed and have put on clean clothing.

"(18) On the outbreak of diphtherial epidemics notification should be given in the public press.

Most important of the recent advances in our knowledge of diphtheria has been the employment of the antitoxin method in its treatment. This method has also been employed for its prophylactic influence. We are indebted to Behring, Katz, Aronson, Surinow, Eastes and others, for our knowledge of this subject. The results obtained by these observers have been of a most encouraging character. During the years 1891, 1892 and 1893, Katz reports that 1,081 cases of diphtheria were treated in the Berlin Hospital for Children, with a mortality of 38.9 per cent., that during 1891 being 32.5 per cent., during the year 1892, 35.4 per cent., and during 1893, 41.7 per cent. From January 1st to March 14th, 1894, eighty-six cases were treated, with a mortality of 41.8 per

cent. Of 128 cases treated since March 14th by the antitoxin method seventeen have died. The percentage of deaths being thus reduced from 41.7 to 13.2 per cent. Unpleasant results of any kind have not been observed to follow the employment of the injections.

The technique is as follows: A syringe holding 5 c. c. should be selected, also one which can be sterilized by means of boiling or an immersion for several hours in a 5 per cent. carbolic acid solution. The region selected for injection should be cleansed with soap and water, and then bathed with an antiseptic solution. Under the age of three years one-half of a cubic centimetre is to be injected for the production of immunity, and double that amount for older children. For the cure of the disease the injections are to be administered during the first two or three days of the attack, giving 2 to 3 c. c. to children under two years of age, 5 c. c. to those between two and ten years of age, and 10 c. c. to older persons. In aggravated cases, after the third day, the amount should be doubled. The injection may be repeated within twelve hours if the first injection is not followed by improvement. Even larger quantities have been advised for unfavorable cases. The evidences of improvement are apparent in from twelve to twenty-four hours, and are pronounced within forty-eight hours. The *Berliner Klinische Wochenschrift*, issue of September, 1894, contains a communication from Behring relating to the serum. He states that serum prepared by Ehrlich and himself is furnished in two forms, and designated as Number 1 and Number 2, the latter being two and one-half times as powerful as Number 1. Number 1 is advised for the treatment of children under ten years of age, providing they are seen by the second or third day of the attack. The dose may require repetition in severe cases occurring in young children, in adults, and in cases which have not received treatment sufficiently early. Number 2 is more efficient for these cases, but owing to difficulties in its production the supply is as yet scant.

Only considerable experience with the antitoxin method can determine its value, but the large mortality from diphtheria, under the best treatment, justifies a prompt consideration of every therapeutic measure which appears rational, and is advanced by competent observers.



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